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Classical reward conditioning of the heart rate response to auditory stimulation in 3 month old infants.

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CLASSICAL REWARD CONDITIONING OF THE HEART RATE RESPONSE TO AUDITORY STIMULATION IN 3 MONTH OLD INFANTS

A Thesis

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Abstract

The present study was conducted in order to determine whether or not HR could be conditioned in 3 month old infants. The conditionability of HR as an autonomic response was viewed in terms of Brackbill and Fitzgerald's (1969) hypothesis regarding differential conditioning results as a function of measuring an autonomic rather than a somatic response. The relative scarcity of conditioning studies measuring autonomic responses prompted a study by Clifton (1970, unpublished data) which yielded inconclusive results. The present study was designed as a replication of the Clifton study with several modifications.

The experiment included two groups of 8 Ss each. The CS was a 14 second 70 dB tone and the UCS a bottle of glucose. Experimental Ss heard the tone for 6 seconds, after which the bottle and tone were presented simultaneously. Control Ss received a Rescorla (1967) control in which the CS and UCS sometimes occurred together and sometimes did not. The results gave unsubstantial evidence of conditioning. No consistent CR was developed and the groups differed on only one of the extinction trials. Several conditions such as satiation, duration and type of CS, and change in state, were considered as possible sources of HR variability which may have obscured the results. Some suggestions were made to modify future infant HR conditioning studies.

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CHAPTER I

INTRODUCTION

At birth the central nervous system of a human infant is still in a very immature state and cortical function is relatively undeveloped (DeKaban, 1959). However, within the first year of life a period of rapid growth occurs during which the myelinization of many of the nerve fibers in the brain takes place, and functions become much more complex (DeKaban, 1959). As the infant develops, his behavior becomes increasingly dependent upon previous learning and less a function of innate reflex systems. Thus, it becomes desirable to acquire an understanding of the infant's capacities early in his development in order to determine what factors are influential and to gain insight into more advanced processes. A second reason for the study of infants, perhaps of even greater importance, is that by establishing early guidelines for normal behavior it then becomes possible to identify behavioral abnormalities at an age when complexity is at a minimum and intervention techniques can be most effectively applied.

The classical conditioning paradigm has served as an important tool in exploring the infant's learning abilities. Although even newborn infants have been found capable of acquiring a conditioned response, not all conditioning experiments have been able to establish conditioning. Soviet investigators (Kasatkin, 1969) have hypothesized that the infant's

conditionability is a function of the type of conditioned stimulus (CS) presented and the infant's maturational level. They believe that some sensory systems are under cortical control at the time of birth and stimuli presented in these modalities can function as effective conditioned stimuli during the first days of life. Vestibular stimuli are considered to be of this type. Other sensory systems, however, are thought to mature at a slower rate, so that if stimuli in these modalities are used as conditioned stimuli only older infants will be able to learn the association (i.e., visual stimuli).

But, even if conditionability does depend on the type of CS presented, which has yet to be validated, the Soviet position may still be inadequate in other respects. A factor which their theory does not consider important in the success of conditioning is the type of response to be conditioned. Kasatkin (1969) implies that if a CS produces successful conditioning with one response, it will also be successful with any other response that can be elicited from the infant. However, recent evidence suggests that contrary to this position, the type of response to be conditioned may be quite important in determining whether or not conditioning will occur. Brackbill and Fitzgerald (1969) report a series of studies in which the conditionability of pupillary dilation (an autonomic response) was compared with the conditionability of an eyeblink (a somatic response). In different experiments conditioning was attempted with a temporal CS, an auditory CS, and a tactile CS. The

success of conditioning was found to be a function of the type of response being measured rather than the type of CS, as proposed by the Soviet theory. Pupil dilation could be conditioned to the temporal CS, but not to the auditory CS or tactile CS. The eyeblink showed exactly the opposite pattern, and could be conditioned to the auditory CS and tactile CS, but not to the temporal CS. Brackbill and Fitzgerald (1969) proposed a tentative distinction between autonomic and somatic reflexes on the basis of their evidence. They suggested that "there must be differential rates of maturation of the cerebral structures that serve as the central portions of conditioned reflex arcs" (p. 203). However, because very few conditioning attempts have been made using an autonomic response, it is not yet possible to determine whether autonomic responses in general affect conditioning differently than somatic responses. It could be the case that pupillary dilation is the only response that yields different results. Additional conditioning studies in which other autonomic responses are measured must be conducted before any conclusions can be drawn. Consequently, it is the purpose of the present experiment to investigate conditioning with a different autonomic reflex, heart rate (HR).

Outline of the Study

The autonomic response which will be the dependent measure in the proposed study is heart rate (HR). HR has recently received considerable attention in the infant literature. In

testing the infant, HR has had the advantage over other psychophysiological measures in that it is relatively easy to obtain and is a sensitive index of the infant's interaction with his environment. Previous studies have devoted much attention to documenting the nature of HR and suggesting different methods of analysis. In addition, HR has been used as the response measure in habituation studies and in research on the orienting response. It is therefore somewhat surprising that only one attempt has been made to condition HR in infants (Clifton, 1970, unpublished data). Although the data from Clifton's study has not yet been fully analyzed, no substantial evidence of conditioning is apparent. But, several methodological difficulties in the study obscure the significance of the results so that no definite conclusions can be drawn regarding the conditionability of HR in infants. In order to clarify this issue, the present experiment was designed as a replication of Clifton's study with several important modifications.

In Clifton's study conditioning was attempted with infants 6 to 12 weeks of age. An 8 sec tone was the CS and a bottle of glucose was the UCS. Each infant received 45 trials which were given during one session. Nine preliminary trials were given to measure the unconditioned HR response (UCR) to the tone alone (6 trials) and to the glucose (3 trials). Next the infant received 30 conditioning trials during which both the tone and glucose were presented. For the experimental Ss the bottle was always offered 6 secs after tone onset, and the infant was

allowed to suck for 8 secs. Control Ss received the bottle for the same duration but it was offered at random times during the trial. The last 6 trials were extinction trials during which the tone was presented but no bottle was offered.

The present study modified this procedure in several ways. To begin with, conditioning was attempted with older infants. The age of the infants in the Clifton study ranged from 6 to 12 weeks. Graham and Jackson (1970) have just recently demonstrated that sometime within the first and third month of life the infant's HR undergoes a developmental shift. For the first few weeks following birth the HR response to any novel stimulus is a pronounced acceleration. But by the beginning of the third month of life it has changed to a primarily deceleratory response. Unfortunately, the subjects in Clifton's study were likely to be within this transition period, making the detection of a clearly defined conditioned response extremely difficult. In order to avoid this problem the infants tested in the present study were from 10 to 14 weeks of age.

A second difficulty encountered by the Clifton study was a rather high rate of subject loss due to either equipment failure, failure of the infants to remain wakeful, or excessive crying. Of 66 Ss tested in the experiment, only 18 remained as subjects in the study. Two changes were made in an attempt to prevent such high subject loss. A sweeter glucose solution was used (20% glucose wt/wt as opposed to 10% glucose used by Clifton). It was hoped that this glucose solution would be

more attractive to the infants and would help equalize differences in drive state. In addition, a shorter conditioning procedure was used. Whereas Clifton's procedure consisted of a total of 45 trials, the present study employed only 30 trials--3 trials to measure the HR response to the bottle alone, 24 conditioning trials, and 3 trials of extinction. This shorter design was intended to reduce subject loss due to drowsiness and crying, yet provide an adequate opportunity for conditioning to take place.

The last modification introduced into the Clifton procedure was an increase in the duration of the tone. In the present study the tone was presented for a total of 14 secs on each trial, as compared with the 8 sec duration used by Clifton. Thus, for the experimental group, the tone was present not only for the 6 sec preceding the bottle and 2 secs in combination with the bottle, but it also remained on for the following 6 secs, during which the baby was allowed to suck on the bottle. This extension of the tone provided a longer pairing of the tone with the bottle of glucose in order to give the infants a better opportunity to establish a connection between these stimuli.

C H A P T E R I I

REVIEW OF THE LITERATURE

Beginning with the experiments conducted by Pavlov in the late 1800's classical conditioning has been widely used in the study of learning. In 1907, N. I. Krasnogorski, one of Pavlov's students, was the first to apply this method to the study of infants. Since that time, numerous classical conditioning studies have been conducted with infants by Russian and American scientists. One of the first problems with which researchers were concerned was documenting the age at which an infant was capable of acquiring a conditioned response. Early studies, performed during the 1930's and 1940's succeeded in conditioning older infants, but were unable to provide conclusive evidence supporting the conditionability of newborn infants (Krasnogorski, 1970; Watson & Raynor, 1920; Denisova & Figurin, 1929; Irwin, 1930; Jones, 1930; Marquis, 1931, 1941; Kantrow, 1937; Wickens & Wickens, 1940; Morgan & Morgan, 1944). More recent experiments have not only conclusively demonstrated conditioning in newborns (Lipsitt, Engen & Kaye, 1963,1964; Lipsitt, Kaye & Bosack, 1965; Kaye, 1965,1967) but have also shown that even premature infants can be conditioned (Polikanina, 1961; Janos, 1965).

Experimenters have also investigated the role played by such parameters as the CS, UCS, and response measure. Soviet researchers have been primarily concerned with manipulating

the type of stimulus employed as the CS (Denisova & Figurin, 1929; Kasatkin, 1960,1969; Kasatkin & Leivakova, 1935). Other investigators have emphasized the importance of the UCS and distinguished between aversive appetitive stimuli in this regard (Lipsitt, 1960; Siqueland, 1970; Brackbill, 1967). The type of response measured has also recently received attention. As pointed out earlier, Brackbill has offered evidence that conditioning may proceed differently depending on whether a somatic or an autonomic response is measured.

Because of the great quantity of research in the area of infant classical conditioning, no attempt will be made to review all of the studies here. Instead only those studies which have particular relevance to the present experiment will be dealt with. Since the primary focus of the present experiment is on the response measure, experiments relevant to this issue will be considered first. Next, studies bearing upon the choice of CS and UCS used in the present experiment will be reviewed.

The HR response. The human HR response to a novel stimulus is usually characterized as either an acceleration or a deceleration, although biphasic or triphasic responses are sometimes observed. Graham and Clifton (1966) in reviewing the HR literature, interpreted the directionality of the response in terms of Sokolov's 1963 model of orienting and defense reflexes. They suggested that the presentation of a low intensity stimulus results in an attending or orienting response (OR) in humans which is accompanied by HR deceleration.

Relatively intense stimuli, however, were thought to produce the defense reflex (DR) of which HR acceleration is a component. As predicted by Sokolov, stimulus repetition was found to result in the rapid habituation of the HR OR, while it did not affect, or in some cases increased, the HR DR. In some of the studies reviewed by Graham and Clifton, a biphasic change in HR occurred following a novel stimulus. This result was most frequently produced by stimuli of moderate intensities, particularly when stimulus onset was sudden. Newborn infants showed a slightly different response pattern to novel stimuli in that the response elicited was always a DR. Very recent research suggests, however, that under optimal conditions, a HR deceleration can be obtained from newborn infants (Clifton, 1971, unpublished data; Malcuit and Clifton, 1971, unpublished data).

The analysis of the HR change to novel stimuli presented by Graham and Clifton (1966) has been further supported by subsequent research. Graham and Jackson (1970) reviewed some of the most current infant studies in this area. In this review, infants of 2½ months and older were generally found to display the typical adult HR orienting and defense reflexes. Infants younger than this age showed HR acceleration even to stimuli which normally produce deceleration in older Ss.

In addition to its effects on the directionality of the HR response, the stimulus presented also influences other aspects of the HR response. Some of the most influential stimulus characteristics include stimulus intensity, stimulus

duration, and the interval between stimulus presentations. The effects of each of these factors will be briefly dealt with.

The magnitude, duration and direction of the HR response are affected by stimulus intensity. In general, as the stimulus intensity increases the magnitude and duration of the HR response increase. As previously mentioned, low intensity stimuli tend to produce HR deceleration, while high intensity stimuli produce HR acceleration. Bartoshuk (1964) investigated the relationship between intensity of auditory stimuli and the HR response. A 1 second 1000 cps tone was presented to newborn infants at four intensity levels ranging from 48.5 dB to 78 dB. A linear relationship was found between HR responses plotted on a log scale and the increase in sound intensity. However, between 62.5 dB and 78 dB the increase in HR response began to decline, thus indicating that somewhere between these two levels the effects of auditory intensity are maximal. Steinschneider, Lipton and Richmond (1966), also working with auditory stimuli, demonstrated that the greater the intensity, the longer the time span between stimulus onset and the peak of the response, and the shorter the latency between stimulus onset and the beginning of the response. The stimuli in this experiment consisted of a 5 sec white noise presented at intensities of 55, 70, 85, and 100 dB. It was also found that as sound intensity increased the HR return level became higher and the time necessary to reach return level increased.

Stimulus duration has also been shown to affect the HR response. Eisenberg (1965) used a white noise as a stimulus and varied the stimulus duration from 200 msec to 1300 msec. She found that stimuli presented for less than 300 msec are most likely not to elicit any response at all. In reviewing Steinschneider's experiment (1966) in which a stimulus of 5 sec duration was used with varying intensities, she concludes that a duration between 1 and 5 sec is probably optimal for white noise. In a much more thorough study, Clifton et al. (1968) investigated stimuli of longer durations (2 sec, 6 sec, 10 sec, 18 sec, and 30 sec). A buzz of 300 pulses per sec was used as the stimulus and was presented at 75 dB against a background of 43 dB. They found that the HR response variation took the form of an inverted U. That is, responses increased up to the 10 sec duration, and for longer stimuli the response size began to diminish. The stimulus of 2 sec duration produced the smallest response, while response variation between the longer stimuli was not as great. Stimulus duration did not affect the response shape or the latency of the response. In commenting on the optimum stimulus duration, Clifton et al. emphasized that it is highly dependent on intensity as explained by the law of reciprocity. This law states that less intense stimuli must be presented for longer durations in order to produce responses equivalent to those evoked by more intense stimuli. Bridger (1961) used even longer stimulus duration (i.e., 40 sec) and offered support for the inverted U described

by Clifton. He found that longer stimuli with short intervals between presentations (½ sec) were the most effective in producing habituation. The longer the duration of the stimulus (past the optimum duration) the less effective it was in producing and sustaining an HR response.

Interstimulus interval (ISI) is also a crucial variable in studies where HR is measured. It is most important that the investigator choose an ISI which is at least somewhat longer than the expected duration of the HR response. This procedure limits the possibility of a response to one trial continuing into the next trial and allows HR to stabilize between responses. There are, however, disadvantages in using too long an interval. Graham, Clifton and Hatton (1968) in using a 75 dB stimulus found 90 sec intervals to be too long. The HR level tended to drop below prestimulus level and the infants were more likely to fall asleep or become fussy. The most frequently used intervals are usually between 20 and 60 secs. Some investigators have abandoned a fixed ISI and have used an index of HR stability (Bartoshuk, 1962; Steinschneider et al., 1966).

Adult HR conditioning studies. The first series of experiments to consistently explore HR conditioning in humans was conducted by Bersh, Notterman and Schoenfeld in the early fifties (Zeaman & Smith, 1965). In these studies, a brief light or tone was employed as the CS. The UCS was a 6 sec shock which followed the CS by six seconds. HR was measured for 2 bpm prior to CS onset and 2 bpm prior to UCS onset. In

general, an HR deceleration was recorded for the two beats prior to the UCS. Using this procedure, Bersh, Notterman and Schoenfeld found that in regard to acquisition, extinction, spontaneous recovery, reacquisition, generalization, and discrimination HR conditioning proceeded in much the same manner as the conditioning of other responses.

However, with continued research it became apparent that HR conditioning was somewhat unique in other respects. One of the most notable peculiarities of HR conditioning was the observation of a CR which differed in direction from the UCR. Whereas the UCR in most studies had been an HR acceleration, the CR was almost always an HR deceleration. This finding was in opposition to the traditional explanation of classical conditioning in which the CR is seen as a weaker version of the UCR.

Much research has been directed toward solving this paradox, and most of the studies conducted can be roughly divided into two categories. The first group of studies was concerned with the possibility that the CR might in actuality be in the same direction as the UCR. It was suggested that some artifactual variable such as respiration might operate on HR in such a way as to mask the observation of the real CR. A second approach considered alternative explanations of HR conditioning not based upon the traditional response substitution view. Because the experiments performed in exploring this question illustrate the typical adult HR conditioning study and comprise most of the recent research, the major studies testing each of

these views are reviewed in the following section.

Respiration was first suggested as a possible factor in HR conditioning by Westcott and Huttenlocher (1961). Their report discussed an unpublished study in which cardiac conditioning was attempted and both HR and respiration were measured. Analysis of their data demonstrated that during pretests 5% of the CS presentations were followed by a sharp intake of breath or gasp and by extinction trials this occurred on 33% of the CS presentations. Elimination of these trials resulted in the absence of conditioned HR acceleration. In the same article they present two experiments which attempt to get at this problem. In the first, respiration was varied along two dimensions, depth and rate, while HR was observed. And, in the second, respiration was paced in time with a metronome, while HR was conditioned. The first experiment showed that changes in HR of up to 30 bpm commonly occurred with slow, deep breathing. In general, the deeper the breathing, the more rapid HR was, with shallow irregular fluctuation. In particular, isolated gasps produced pronounced and consistent biphasic HR responses. The authors cite an example of the typical HR response to a gasp in which HR changed from a prestimulus level of 83 bmp to 95 bpm and then decelerated to 70 bpm after which it returned to prestimulus level.

The second experiment was a classical conditioning procedure in which the subjects were instructed to name colors off a color wheel until a buzzer sounded, whereupon they were to breathe

in time with a metronome (very rapid 46 breaths per minute), until the buzzer went off (7 seconds duration). The offset of the buzzer was followed by a shock during conditioning trials. The shock level used was very intense, the highest the subjects were able to tolerate, and according to the author, generally, produced gross bodily movement. Subjects received a number of practice trials, 10 adaptation trials (buzzer alone), followed by 10 conditioning trials. HR analysis included the mean HR for 7 prestimulus seconds, the mean HR for 7 post-stimulus seconds, and a second by second analysis in order to determine the form of the HR wave. Results indicated that the metronome was successful in controlling respiration and that no significant changes occurred over trials. During adaptation trials the mean HR response was a 1.1 bmp deceleration. During conditioning trials the mean HR response was a 2.9 bmp acceleration (average of last three conditioning trials). The form of the HR responses they observed was a marked acceleration which had fallen to near prestimulus level by the 7th second (see Fig. 1A). These authors conclude that HR conditioning is not a result of respiration and that when respiration is not controlled it is analogous to superimposing the HR response to a gasp upon the HR conditioned response. The result would be a smaller acceleration followed by a pronounced deceleration. But, when respiration is controlled the real nature of the conditioned response is evident that it is completely acceleratory. This evidence, however, is contrary to that which they cite in the

unpublished study where elimination of trials upon which a gasp occurred in absence of an acceleration.

Because of his discrepancy Wood and Obrist (1964) attempted a similar study to determine the real nature of the conditioned HR response. They hypothesized that the conditioned HR would be a deceleration because it would result from vasomotor activity, independent of respiration. They utilized two respiratory control groups, one in which respiration was paced at a normal rate to the onset and offset of a light (controlled respiration), and another in which respiration was not controlled (uncontrolled respiration). The level of shock to be administered was determined in the same manner as that used by Westcott and Huttenlocher. However, it was not judged to be as intense and did not produce gross bodily movement. Ten adaptation trials were given in which two tones of different frequencies (1000 cps and 500 cps) were presented five times each. The tones were of one second duration with variable intertrial intervals and were counterbalanced across subjects as to which served as CS+ and CS-. Then, five blocks of 10 conditioning trials were given. In each 10 trial block the subjects randomly received 6 trials of CS+ where the tone was followed after 6 seconds by the shock, 2 trials of CS+ where no shock followed the tone (test trials), and 2 trials of CS-. Analysis of the results indicated that when respiration was not controlled it increased in amplitude following the CS+ and a biphasic HR occurred. When respiration was controlled no significant HR acceleration occurred, and

deceleration was attenuated (see Fig. 1B). Wood and Obrist reconciled their results with those of Westcott and Huttenlocher by pointing out that Westcott and Huttenlocher only measured the HR response for 7 seconds and if they had used test trials and measured HR for a longer period of time they almost certainly would have found a deceleratory component in the response since their last measured showed HR to be decelerating. They further suggest that the shock used by Westcott and Huttenlocher was much more intense and could account for the large acceleration these authors found.

Headrick and Graham (1969) pointed out that Wood and Obrist did not use the same respiratory control as did Westcott and Huttenlocher. The former authors employed a respiration control at normal respiratory rates while the latter authors used a much more rapid rate (46 breaths per minute). This difference could account for the marked acceleration found by Westcott and Huttenlocher even though respiration had been controlled. In order to determine if this were the case Headrick and Graham (1969) replicated the Wood and Obrist prodedure with the addition of a third respiratory control group at the same rapid rate used by Westcott and Huttenlocher. Results of this study showed that the uncontrolled respiration group tended to breathe more slowly following tone onset and more deeply on CS+ trials, the latter effect becoming more pronounded across trials. The other two groups maintained a consistent respiratory pattern except that the rapid respiration group tended to breathe a little

more deeply on CS+ trials but with no change over trials. However, in spite of the differences in respiration between the groups, they were found to differ very little with respect to the conditioned HR (see Fig. 1C). No significant differences were found between the uncontrolled respiration group and the group in which respiration had been controlled at a normal rate. The rapid respiration group differed from the other two groups in that the deceleratory component of the response was greater during adaptation trials and this persisted into the conditioning trials, being most evident on CS- trials. In all three groups, the response was triphasic beginning with a slight deceleration (approximately 1 bpm) followed by a slight acceleration (1-2 bpm), and then a large deceleration (4 bpm) which was maintained until UCS onset or scheduled onset.

Headrick and Graham conclude that the large shock given by Westcott and Huttenlocher probably accounts for the acceleration found. They suggest that in other instances where acceleration was found, it might have been produced by over demanding respiratory maneuvers. They reject an explanation previously offered that deceleration might be due to an OR produced by absence of expected shock because the deceleration occurs before the shock is due to occur. Nor do they believe that the deceleration is an unhabituated portion of the initial CR to the tone since its form is quite different from the response which occurs during adaptation trials. These authors offer the explanation that the initial deceleration is an OR to the tone, the acceleration

results from a preparation for action dependent upon the motivating and energizing factors in the particular experiment, including the degree of shock anticipated. The large deceleration is seen as the portion of the response subject to conditioning and represents an anticipatory OR.

Investigators in different laboratories conducted studies testing other hypotheses about the source of the deceleratory CR. Zeaman and Smith (1965) reviewed a large number of studies directed toward this problem. Three alternatives they considered were stimulus substitution, mediation, and drive reduction. The first study performed by Zeaman et al. in 1954 involved the use of a brief auditory CS and a 6 second shock UCS. The CR was biphasic and at first appeared to be determined by drive reduction. Whatever the heart was doing at the time the shock terminated would be reinforced and subsequently occur in response to the CS. This theory was tested in a second experiment which employed a shock so short that the heart did not have time to react before the shock was terminated and a shock so long that the heart had finished reacting at the time of shock termination. The drive reduction theory predicted no CR under these conditions since the heart was not reacting at the time of shock termination. However, results indicated that conditioning occurred in both instances, necessitating the rejection of the drive reduction hypothesis.

An alternative explanation proposed was that fear mediated by the tone resulted in HR deceleration. This theory was tested

by utilizing a long delay period during which numbers were presented successively in an increasing order up to the time of expected shock. Using this procedure the HR response during number presentation on the first trial was a gradual acceleration up until immediately prior to shock presentation when a sharp deceleration occurred. On trials after the shock had been experienced the initial acceleration disappeared. In other experiments a noxious noise and an extremely painful shock were used as the UCS. When the noise was the UCS the accelleratory component usually present on the first trial was absent. However, the HR deceleration which had preceded shock onset did not disappear and still occurred immediately prior to noise onset. When the UCS was a very painful shock, the acceleratory component appeared not only on the first trial, but on subsequent trials as well. On the basis of this evidence, Zeaman et al. hypothesized that HR acceleration was a result of a conditioned emotional response to anxiety and that HR deceleration was a conditioned emotional response to fear. Thus, little anxiety would be produced by the threat of a noise so that no HR acceleration would be expected in this instance. A moderate shock would create anxiety only on the first trial, after which Ss would no longer be anxious since the shock would be less painful than expected. Finally a strong shock would result in anxiety on the first trial and on subsequent trials so that HR acceleration would be present on all of the trials. In all of these instances, however, the HR deceleration prior to UCS onset

would continue to occur since the S would be apprehensive about the presentation of a noxious stimulus.

Unfortunately, this mediation hypothesis ran into difficulty when an experiment was conducted in which the UCS was a pleasant sound. The hypothesis predicted that under these circumstances there would be no apprehension and, therefore, no HR deceleration. But the results showed that HR deceleration still occurred. Consequently, Zeaman et al. were forced to alter the mediation theory to the effect that HR deceleration occurred as a result of a state of simple anticipation or attention rather than only to fear.

Finally, stimulus substitution remained as a possible explanation. Studies conducted by these authors using sustained inspiration and sustained expiration as respiratory controls found the CR to be a large acceleration without a deceleratory component. Since this response was highly similar to the HR response produced by the UCS, a stimulus substitution theory could not be ruled out. Zeaman et al. hypothesized that the CR is basically an HR acceleration produced by anxiety and that respiratory changes accompanying anticipation or attention could account for the HR deceleration. It should be pointed out, however, that this theory requires respiratory interference to be extremely consistent in order to produce the characteristic HR deceleration normally observed during conditioning experiments. Other authors have subsequently noted that sustained inspiration or expiration is inadequate as a respiratory control in that it

creates conditions quite different from those present during an HR conditioning experiment (Hastings and Obrist, 1967). Moreover, when respiration is paced, as in experiments previously discussed, the deceleratory component is still present.

Hastings and Obrist (1967) also investigated several hypotheses which might explain the occurrence of a deceleratory CR. The possibilities they considered were: 1) the HR deceleration is actually a CR although in the opposite direction; 2) the HR deceleration is an orienting response; 3) the HR deceleration is a previously conditioned response mediated through anticipation during the interstimulus interval.

To test these hypotheses Hastings and Obrist conducted an experiment in which three different CS-UCS intervals were used. These were .8 seconds, 7 seconds, and 13 seconds. According to the authors each of the three hypotheses predicted a different result under these conditions. If the HR deceleration was a CR as suggested in the first hypothesis, the HR response would be the largest when an optimal ISI of .8 seconds was used. Hypothesis 2, that the HR deceleration was an orienting response, predicted that the length of ISI would have no effect on the size of response. The third hypothesis, that a previously conditioned CR appeared in anticipation, predicted that the peak of the response would occur just prior to UCS onset regardless of ISI. In addition, the response would be smallest for the .8 second ISI since less time would be available for anticipation.

The *S*s in the experiment were 45 male college students.

They were divided into three groups according to ISI. For all Ss the UCS was a shock. Ss were given a series of 6 second shocks ascending in intensity until they reached a shock level they considered intense or until a level of 1.5 mA was reached. This determined the shock level to be used as the UCS. Respiration was paced for all Ss at their normal respiratory rate using an auditory stimulus which simulated breathing. The Ss were given 10 adaptation trials in which the 2 stimuli to be used as CSs (a red light and a blue light) were each randomly presented 5 times. Conditioning consisted of 36 trials. The CS+ was presented 26 times and followed by shock on 16 of these occasions. The CS- was presented 10 times and never followed by shock. HR was measured for every second beginning 1 second prior to CS onset and continuing for 25 seconds.

The results supported the third hypothesis. The deceleration was very small for the .8 second ISI and large, peaking just prior to UCS onset when the ISI was 13 seconds. The response was present on the first trial rather than developing with successive pairings of CS and UCS, as would be expected if the HR deceleration were a CR.

A final study which has also dealt with the problem of what is conditioned in HR conditioning was performed by Wilson (1969). In this study, Wilson manipulated five different variables and observed their effects on the conditioning process. The variables were CS duration, trace versus delay conditioning, the administration of habituation trials prior to training

versus following training, intermixing habituation trials with training trials versus presenting them in blocks, and type of UCS. The design consisted of 12 separate groups each composed of 16 college students. For 11 of the groups the UCS was a shock averaging 2.4 mA, and for the last group a 2 sec noxious noise was the UCS. The CS was always a 1500 Hz tone presented at 70 dB through earphones. The CS duration varied for different groups and was 1 sec, 5 sec, or 10 seconds. For groups which received the delay conditioning procedure the shock occurred immediately following the CS. Trace conditioning groups received either a 1 second CS followed 4 seconds later by shock or a 1 second CS followed 9 seconds later by shock. Forty-five trials were presented at constant 30 second intervals. HR was measured by using a leg cuff which was alternately inflated for 3 minutes while HR was recorded, then deflated for 2 minutes.

On the basis of the results of this experiment, Wilson suggested 2 factors as influential in determining the direction and form of the CR. He offered evidence that the CR which develops is a composite first of the original response to the CS, and second of an attending response which arises from the predictability of the situation. The original response to the CS in thisexperiment was always a biphasic response of HR acceleration followed by HR deceleration. Although the response was always of the same shape, its timing varied depdnding on the duration of the CS. The conditioned portion of the response

which arose as a consequence of the predictability of the situation was an HR deceleration at the time of expected shock. The CR which developed was found to be a combination of the exact timing of the HR response to the tone, depending on its duration, and the HR deceleration which reflected anticipation of the shock. For the CS of 5 sec duration, the CR was the most pronounced. Under this condition, the HR deceleration to the CS coincided with the HR deceleration produced by anticipation to produce a large deceleration. For the 1 sec and 10 sec CSs the two components of the CR were out of phase causing the overall response to be a much smaller deceleration.

This conclusion differs only slightly from that arrived at by Hastings and Obrist. Wilson simply places more emphasis on the response to the CS as an important part of the CR. In these two studies as well as those conducted by Zeaman et al. the conditioned segment of the response is viewed as an HR deceleration produced by expectancy or anticipation.

HR conditioning in infants. With the exception of the Clifton study, described in detail on page 4, only two studies have attempted classical conditioning with infants while measuring HR. Polikanina (1961) conducted a study with premature infants comparing the conditionability of autonomic and somatic components of a reflex reaction to the smell of ammonia. The conditioned stimulus was a tone presented for 3 to 5 seconds at the beginning of conditioning and gradually extended to a duration of 10 to 15 seconds. The autonomic components of the

conditioned response were HR acceleration and slowing or arrest of respiration. The somatic response included various motor activities such as grimaces, blinks, and sucking or swallowing reactions. Infants received 6-7 pairings of tone and odor every day beginning soon after birth, until a stable conditioned reaction was established. The number of pairings necessary to establish conditioning varied for the different response components. Respiration was most rapidly established, followed by the motor response, and finally HR. The degree of prematurity also affected the rate of conditioning. The greater the prematurity the longer it took to establish conditioning.

Although this study was well performed, no indication is given of the method of determining the HR response. In addition, a control group in which infants received only the CS would have been desirable. Nevertheless, this study offers convincing evidence that various reflexes differ in their susceptibility to conditioning during early infancy. It is not surprising that respiration was the most easily conditioned response in this experiment in view of the nature of the UCS (ammonia odor). It appears likely that the response measure most intimately related to the stimuli presented will be the most susceptible to conditioning.

Lipsitt and Ambrose (SRCD paper, 1967) demonstrated successful temporal conditioning of autonomic responses in newborn infants. Three different stimuli were used as CSs including the sound made by dropping a wooden ball on a table, the presentation

of a strong anise odor, and vestibular stimulation given by rocking the infant in a special apparatus. Eleven successive trials were given for each stimulus. For the first 8 trials the stimulus was presented at a constant 30 sec interval. The last 3 trials were mock trials on which the 30 sec interval was marked but the stimulus was not presented. Each mock trial consisted of a 5 sec period at the usual time of stimulus presentation. Another 5 sec period which occurred 15 sec after the usual time of stimulus presentation was used as a control comparison. Three autonomic response measures were recorded during the mock trials and control periods. These were HR, respiration and motility. A change in HR of 2 bpm or more was recorded as a response. Two independent judges scored respiration and motility responses. Each infant went through 9 mock trials (3 for each of the 3 stimuli) and therefore had 9 opportunities to respond.

The average number of responses made was 4.53 on the mock trials and 2.46 during the control period. Although the number of responses was significantly greater on the mock trials than during the control period ($p = .01$), it is evident that temporal conditioning was only weakly established. Since an average of 2.43 responses occurred by chance, only 2.10 responses of the group average can be attributed to temporal conditioning, out of the 9 opportunities. Since the data was only in a preliminary state of analysis, no mention was made as to which response measures showed the greatest susceptibility to conditioning.

Because a change in any one of the 3 response measures would be recorded as a response on a particular trial, it is possible that responses were randomly distributed among the three different response measures and no single response modality showed substantial evidence of conditioning. Until the final analyses have been performed on these data, no conclusions should be drawn concerning the conditionability of HR from this experiment.

Conditioning of other autonomic responses in infants. The fact that so few infant conditioning studies have used HR as the response measure is not so surprising when one considers the scarcity of conditioning studies in which any autonomic response was used as the response measure with infants. Moreover, the information derived from those studies is even further reduced because methodological problems have made it difficult to interpret them.

Jones (1928,1930) was one of the earliest experimenters to attempt autonomic conditioning in young infants. Using GSR as the response measure he conducted experiments with 3 infants who ranged from 3 to 6 months of age at the beginning of the procedure. The UCS was described as an inductorium which produced a mild electrical current that the infants seemed to enjoy. Several different stimuli functioned as the CSs including a light, a touch, and several different sounds. For all 3 infants, conditioning was established within 14 associations of the CS and UCS. In addition, the response was extinguished but showed

spontaneous recovery on successive days. Unfortunately, the small number of Ss and lack of statistical tests prevent any definite conclusions from being drawn. Moreover, the results are somewhat unusual in view of the fact that many investigators have experienced considerable difficulty in eliciting a reliable GSR in infants.

Krachkovskaia (1959) showed that young infants could acquire a conditioned leukocyte rise in anticipation of feeding. Leukocytosis is an increase in white blood cell count which occurs during the process of digestion. Normally, during the first few days of life, leukocytosis becomes a stable response after the ingestion of food. Through careful observation, Krachkovskaia found that when infants were fed on a regular 3 hour schedule, leukocytosis gradually occurred both in anticipation of food and following food and following food intake. An experiment was conducted with 3 children, 1 month of age or older. These infants all showed anticipatory leukocytosis continued to occur as on the 3 hour schedule. But, on the second day, after 8 feedings on the new schedule had occurred, leukocytosis no longer appeared at 3 hour intervals, but anticipated the 4 hour feeding.

Although the phenomena described by this study is quite interesting the results should be regarded with caution. Only 3 infants were observed during change in feeding schedule and no statistical tests were conducted. Furthermore, a control group in which infants were switched from a 4 hour schedule to

a 3 hour schedule would have been desirable.

Brackbill and Fitzgerald (1969) reviewed a series of studies in which pupillary dilation and constriction were the conditioned responses. What makes these experiments even more interesting is the fact that parallel experiments were conducted in which a somatic response, the eyeblink, was the conditioned response. Three sets of experiments were performed, each using a different kind of CS. These included a temporal stimulus, an auditory stimulus, and a tactile stimulus. Two conditioning experiments were attempted with each of these stimuli. In one the pupillary response was measured and in the second the eyeblink was the conditioned response.

Brackbill, Lintz and Fitzgerald (1968) report the results of the pupillary and eyeblink experiments using a temporal CS. Sixteen infants, approximately 2 months of age, participated in the pupillary conditioning experiment. The UCS was a 4 second change in illumination produced by turning a bright light on or off. Pupillary dilation was measured in half of the Ss, for whom the change in illumination was turning the light off. For the remaining Ss, the onset of the light was the UCS and pupillary constriction was measured. The CS was 20 seconds of elapsed time. Thirty-two conditioning trials were given during a single session. Of these 9 trials were probe trials during which the UCS was not presented. Following conditioning, 35 extinction trials were given. Control Ss followed the same procedure except that the intertrial interval varied randomly from 10 to

30 seconds for this group. To test for conditioning on probe trials, a 5 second interval beginning 2.5 seconds prior to the expected CS was compared with a neutral 5 second interval. The results indicated successful conditioning for every infant in the experimental group. For Ss showing pupillary dilation, the average pupillary size was 2.54 mm at the time the CS was expected and 2.43 mm during the neutral interval. For the pupillary constriction group the average pupillary size was 2.57 mm when the CS was expected and 2.88 mm during the neutral period.

Temporal conditioning was also attempted with the eyeblink response. In this experiment the UCS was a puff of air to the eye. The Ss in the experiment were 8 infants of approximately 2 months of age. The experiment was conducted during 2 sessions on consecutive days so that the infants could serve as their own controls. The first session was used as a control for pseudoconditioning. The infants received 32 trials at random intervals of 10 to 30 seconds. Eight trials were probe trials on which the UCS was omitted. During the second session, conditioning was begun and trials occurred at constant 20 second intervals. The infants were given as many conditioning trials as they would tolerate with the average number of trials being 82. Probe trials were randomly interspersed among the conditioning trials at a ratio of 1 to 3.

The analysis of the results indicated that the number of eyeblinks was evenly distributed throughout the duration of

the probe trials and not concentrated around the 20th sec as would be expected if conditioning had occurred. Consequently, the authors concluded that conditioning had not been demonstrated.

Brackbill, Lintz and Fitzgerald (1968) described a second set of conditioning experiments in which the CS was a sound. As in the first experiment, conditioning was attempted with pupillary dilation and constriction. The UCS was a change in illumination as previously described in the temporal conditioning experiment. The CS was a complex sound of 5.5 sec duration. For experimental Ss, the sound began 1.5 sec prior to UCS onset. For control Ss, the CS and UCS began simultaneously. Intertrial intervals varied between 10 and 30 sec for both groups. Thirty-two conditioning trials were given, 9 of which were probe trials. Comparisons were made in which a neutral 5 sec interval preceding CS onset on probe trials was compared with the 5 sec interval following CS onset. The results showed no evidence of conditioning. For Ss for whom pupillary dilation had been the CR the average pupil size was 2.59 mm during the neutral 5 sec interval and 2.61 mm during the 5 seconds following CS onset. The pupillary constriction Ss showed an average pupillary size of 2.24 mm during the neutral period on probe trials and 2.25 mm following CS presentation.

The second study, however, which involved eyeblink conditioning was able to demonstrate successful conditioning. In this study the CS was a tape recorded sound of .2 sec duration

presented 1 sec prior to the UCS, a puff of air to the eye. The intertrial interval varied from 30 to 60 seconds. Twenty infants, approximately 2 months of age, served as Ss in the experiment. During conditioning the 8 Ss in the experimental group received up to 25 trials during each daily session. Probe trials were randomly interspersed among conditioning trials in a ratio of 1 to 3.5. Sessions were conducted until a strict criterion of conditioning had been met. Infants were required to give eyeblinks on 9 out of 10 successive trials. All infants showed successful conditioning. The number of trials necessary to reach criterion varied from 50 to 274 trials.

The 12 infants who served as controls were divided into 3 groups. The infants in the major control group received the same number of UCS and CS presentations as the 4 slowest learning experimental Ss, but in a random unpaired order. The second group of 4 control Ss received a single session of trials during which the CS alone was presented at random intervals. And the last control group, which provided a measure of spontaneous blinking, received a single session of no stimulation. No evidence of conditioning was present in any of the control groups.

In summarizing these last two studies Brackbill and Fitzgerald (1969) make the obvious point that infants in the eyeblink experiment received many more trials than infants in the pupillary conditioning experiment. However, they report that one infant in the pupillary conditioning experiment was given

an additional 360 trials and still gave no evidence of any conditioning. Nevertheless, it is unfortunate that additional trials were not given to all of the Ss in the pupillary experiment to conclusively demonstrate that lack of conditioning was not a result of too few trials.

Fitzgerald and Brackbill (1971) attempted pupillary and eyeblink conditioning to a tactile stimulus. The CS was produced by lightly stroking the infant's cheek with a nylon monofilament for 2 seconds. In the first experiment the response measured was an eyeblink elicited by a puff of air to the eye. The interstimulus interval was 1 sec and the intertrial interval varied between 10 and 30 sec. The Ss were 14 infants with a median age of 36.5 days. The 10 Ss in the experimental groups received 6 sessions of trials conducted during the mornings and afternoons of 3 consecutive days. On the first session, the tactile stimulation was presented alone 41 times to produce habituation. The next 4 sessions consisted of 32 conditioning trials each. On 9 of these 32 trials the UCS was omitted to test for the presence of conditioning. Extinction was given during two sessions each consisting of 41 trials on which the CS was presented alone. The 4 Ss in the control group followed the same procedure except that during conditioning trials the CS and UCS were randomly presented in an unpaired order.

To test for conditioning the number of eyeblinks during a 5 sec interval following CS onset on probe trials was compared with the number of blinks during an equal interval prior to CS

onset. The results showed substantial evidence of eyeblink conditioning. During the interval prior to CS onset an average of only 3.88 eyeblinks occurred on probe trials. While during the interval following CS onset an average of 19.11 blinks were recorded. This difference was statistically significant with a $p = .01$.

In the second experiment reported by Fitzgerald and Brackbill (1971), pupillary conditioning was attempted with the same tactile CS. Both pupillary dilation and constriction were measured. The UCS was a 4 sec change in illumination. As in the eyeblink experiment the interstimulus interval varied from 10 to 30 seconds. The Ss were 24 infants with a median age of 41.5 days. Sixteen infants were in the experimental group. The experimental Ss received an average of 282 conditioning trials over 5 sessions. Of these an average of 79 were probe trials randomly intermixed among the other trials in a ratio of 3.5 to 1. Half of the Ss in the control group received one session during which the CS and UCS were presented randomly and unpaired. The remainder of the control Ss received 164 presentations of the tactile stimulus alone. Comparisons were made between responses for 5 seconds prior to CS onset and responses during the 5 seconds following CS onset on probe trials. The results indicated that no conditioning had occurred.

In summary, the experiments by Brackbill and her colleagues have demonstrated successful conditioning of change in pupillary diameter to a temporal CS, but not to an auditory CS or tactile

CS. The eyeblink response, on the other hand, was successfully conditioned to an auditory CS and a tactile CS, but not to a temporal CS. In interpreting these results, it should be pointed out that pupillary conditioning is somewhat of a controversial issue. Pupillary conditioning has been difficult to establish in adults except when an aversive UCS such as shock was used (Kimble, 1961). For this reason it has been suggested that it may occur as part of a generalized emotional reaction. Young (1965) noted that the lack of feedback during a pupillary response should make conditioning impossible. Thus, pupillary conditioning may constitute a unique instance of autonomic conditioning.

The use of glucose as the UCS. The widespread success of appetitive stimuli as UCSs in classical conditioning experiments suggested the use of an appetitive stimulus as the UCS in the present experiment. Although conditioning has been demonstrated with both appetitive and aversive UCSs, young infants appear to condition more easily when an appetitive UCS is used (Lipsitt, 1960; Siqueland, 1970; Brackbill, 1967). In fact, very young infants, less than 2 months of age, are extremely resistant to conditioning procedures in which an aversive UCS is used. Older infants will condition to mildly aversive stimuli such as a puff of air to the eye or a bright light, but are more difficult to condition when a stronger stimulus, such as a shock to the foot, is used. For these reasons almost all classical conditioning studies conducted with newborn infants, and many

of the studies conducted with older infants, have utilized an appetitive UCS. Most frequently, the UCS has been the infant's bottle of formula, although a bottle of glucose, placement in the feeding position, and a rubber nipple have also been used.

Of the many studies performed using an appetitive UCS, only one study appears in the literature in which conditioning was not established. In this study, Abrahamson (1970) attempted to temporally condition sucking in newborn infants using a bottle of formula as the UCS. After 160 stimulus pairings no evidence of conditioning was apparent. However, following a similar procedure the same author successfully obtained conditioning when the temporal CS was replaced with an auditory CS. Thus, the CS rather than the UCS was apparently the determining factor in the failure to establish a conditioned response.

Of the several stimuli possible as UCSs in the present experiment, a glucose solution appeared to be the most appropriate. One reason for this conclusion was that glucose was the UCS in Clifton's experiment, which the present study attempted to replicate. Secondly, a sweet glucose solution was considered to be attractive to most infants, even if they were not particularly hungry. This assumption was supported by a series of head turning studies which demonstrated that glucose solution could function effectively as a reinforcer (Siqueland and Lipsitt, 1966). In these studies the infants learned to turn their heads to receive a 5% glucose solution.

Glucose has previously been used as the UCS in one other

classical conditioning study and successful conditioning was obtained. Lipsitt, Kaye and Bosack (1965) tested 20 newborn infants using for the CS a flexible rubber tube through which the UCS, a 5% glucose solution, was delivered. The 10 experimental Ss sucked on the tube for a total of 15 seconds. During the last 5 seconds these infants were given glucose through the tube. Control Ss were also allowed to suck on the tube but the glucose was given with a syringe 30 seconds after the tube had been removed from the infant's mouth. All Ss received 6 base trials of tube alone, 10 conditioning trials, 10 extinction trials, 5 reconditioning trials, and 5 more extinction trials. The two groups were compared for amount of sucking during the first 10 sec of tube presentation. The experimental Ss were found to increase the number of sucks during the conditioning trials and differed significantly from the control Ss who decreased in number of sucks. During the first set of extinction trials experimental Ss dropped in number of sucks until they were at the same level as control Ss. Then, the number of sucks rose again for experimental Ss during the reconditioning trials and then finally decreased once again during the last extinction trials. Thus, this experiment also suggested that glucose could be used effectively as the UCS to establish classical conditioning.

The use of a tone as the CS. Several important considerations suggested the use of an auditory stimulus as the CS. First of all, the HR response to sound has been well documented in

both infants and adults (i.e., Richmond et al., 1966; Graham et al.; 1968; Lipton et al., 1966). In addition the HR conditioning studies which have been conducted have almost exclusively employed an auditory stimulus as the CS (Westcott & Huttenlocher, 1961; Zeaman & Smith, 1965; Wilson, 1969). One of the reasons for the strong popularity of auditory stimulation is that it is easy to control and can be systematically varied while other parameters are held constant. Moreover, in infant studies auditory stimulation has had the advantage over other stimulus modalities such as vision in that the infant is likely to perceive the stimulus even when drowsy or attending to something else. A final factor influencing the choice of an auditory stimulus was the precedent set in the Clifton study, in which the CS was a tone. Thus, for these reasons, an auditory stimulus was employed as the CS in the present study.

The particular stimulus used was determined by several additional factors. The tone frequency was chosen to fall within the range of human voice frequency. Eisenberg (1965) suggested that infants are most receptive to stimulation well under 4000 cps. A pure tone was used rather than a complex tone because of practical considerations of equipment availability. Brackbill (1967) discusses the relative effectiveness of simple and complex tones and concludes there is no strong evidence that one type of tone is preferable to another.

The duration of the tone in the present experiment was 14 seconds. This interval allowed for 6 seconds of HR response to

the tone alone and in the experimental group an additional 8 seconds of pairing the tone to the bottle. This duration was very close to the 10 second duration which was found to be most effective in eliciting an HR response in Clifton et al. (1968). The intensity of the tone was 70 dB. Previous investigators have concluded that intensity levels between 62.5 dB and 78 dB are preferable in infant research (Eisenberg, 1965; Bartoshuk, 1964).

C H A P T E R I I I

METHODOLOGY

Subjects. Thirty one infants participated in the study. Of these 15 infants were eliminated for the following reasons--7 for crying, 5 because of lost data due to movement artifacts; 2 because of equipment failure, and 1 for falling asleep. Data analysis was carried out on the remaining 16 Ss, 8 of whom were in the experimental group and 8 in the control group. These infants ranged in age from 74 to 96 days at the time of testing. The experimental group was composed of 3 girls and 5 boys whose average age was 83 days. The control group contained 2 girls and 6 boys with a mean age of 81 days.

Apparatus. A BRS Digibit System controlled the onset, offset, and duration of the stimuli, and the intertrial interval. A tone of 1300 cps was produced by a digibit audio oscillator. The tone was presented through a speaker located approximately 8 feet from the infant and at an intensity of 70 dB at the site of the infant's ears (background noise level was measured at about 48 dB). The EKG was recorded from three chest leads and monitored by a Hewlett Packard polygraph. A single pulse for each heart beat was recorded on channel 1 of a Revox tape recorder, which simultaneously marked the onset of CS presentation on channel 2. The average inter-heartbeat-intervals (interval between R waves) for one prestimulus second and 18 post-stimulus seconds were punched out on cards in milliseconds

by a PDP8/I computer. Further statistical analyses were performed using a CDC 3600 computer.

Procedure. The names of subjects within the eligible age range were obtained from the town hall. Mothers were contacted by telephone and offered $5 for bringing their infants to the infant laboratory located at the University of Massachusetts. Infants were randomly assigned to the experimental group and control group. When the mother arrived, the experimental procedures were briefly explained to her and information concerning the infant's feeding habits was obtained. Electrodes were attached to the infant's chest and he was placed on his back in a semi-reclining position in an infant seat situated in a crib. One experimenter remained in the room at the end of the crib, out of the infant's sight, to administer the bottle of glucose. A dim light, not visible to the infant, signaled the assistant when to offer and withdraw the bottle. A second experimenter monitored the recording equipment in an adjoining room. The infant's mother was also in the adjoining room from which she could observe her baby through a one-way mirror.

Design. For each infant trials 1-3 consisted of the bottle alone. These trials were included to provide an index of the infant's initial response to the bottle and give him an opportunity to become used to it before conditioning began. Trials 4-27 were conditioning trials. Infants in the experimental group received a 30% sugar glucose solution 6 sec after the onset of the tone during these trials. The tone and bottle

were then present together for an additional 8 secs, terminating simultaneously. Infants in the control group heard the tone at the same intervals as the experimental group. However, the presentation of the glucose occurred randomly during the interval between one tone onset and the next tone onset, sometimes occurring in conjunction with the tone, and sometimes not (Rescorla, 1967 control). For all infants trials 28-30 were extinction trials. The tone was presented, but the bottle was not offered. The intertrial interval varied randomly between 31 and 41 secs from the onset of one tone to the onset of the next tone. This interval allowed for at least 19 secs from bottle to the next tone onset.

Data reduction. For every subject the data on each trial was converted from interbeat intervals to beats per minute (BPM). A measurement of HR in BPM was obtained for each second beginning with 6 prestimulus seconds and continuing for 18 secs following stimulus onset, for every trial. For each second the average HR in milliseconds was determined by weighting heartbeats according to proportion of the one second interval they comprised. Thus a heartbeat only partially occurring with the one second interval would be weighted less than a beat occurring completely within the interval. A CDC 3600 computer was used for further data analysis. Trials were averaged into 10 three trial blocks of 19 seconds for each S. Group averages were obtained for single trials and for the three trial blocks over seconds.

C H A P T E R I V

RESULTS

Test for LIV. Before testing the data for evidence of conditioning, it was necessary to determine whether the law of initial value (LIV) was influencing the results, and if so, to correct for it. LIV refers to the homeostatic process in which autonomic activity is maintained within an optimum range. When the prestimulus level of functioning is already higher or lower than base level, homeostatic controls prevent the system from fully responding to stimuli which would further increase the deviation from base level (Sternback, 1966). Thus, on trials where prestimulus HR is higher than normal LIV would reduce the effect of stimuli which resulted in HR acceleration. Similarly, on trials where HR is lower than base level LIV would reduce the effect of stimuli which resulted in HR deceleration.

To test for the presence of LIV a measure of the prestimulus level was correlated with the HR response to the tone. The prestimulus score used in the correlation was the average HR of the 6 seconds immediately preceding tone onset. The score used as the HR response to the tone took into consideration both the magnitude and direction of the HR response. It was arrived at by finding the mean HR during the 6 seconds immediately following tone onset and subtracting it from the mean of the 6 prestimulus seconds already calculated, to obtain a positive or negative difference score. The prestimulus scores were then

correlated with the difference scores for all Ss on all trials where the tone was presented. The correlation obtained for Ss in the experimental group was r = .11 (210 observations) and for Ss in the control group r = .26 (216 observations).[1] These results did not provide evidence for the presence of LIV in the data since if LIV had been operating, a negative correlation would have been obtained. Consequently, the data were not corrected for LIV effects.

The HR response to the UCS and CS. The initial response to the UCS (bottle of glucose) was recorded during the first three trials. The responses for both the experimental and control groups are shown in Figure 2. Both groups responded with a 3-4 BPM acceleration which was maintained until the UCS was terminated.

The initial response to the CS (tone) was measured during trials 4-6. During these trials the experimental Ss were offered the bottle 6 seconds after tone onset. Thus, the two groups received comparable stimulation only during the first 6 seconds of CS presentation. The responses made by both groups are presented in Figure 3. For both groups the HR response during the first 6 seconds of CS presentation was a deceleration. The experimental group decelerated approximately 4 BPM and the control group decelerated 2.4 BPM. Following the sixth sec of CS presentation the Ss in the experimental group received the

[1] Two subjects were missing the 3 extinction trials (see Appendix B).

bottle of glucose which resulted in HR acceleration. The control Ss, however, who continued to hear the tone without having the bottle, showed further HR deceleration until the eighth sec for a total response of 5.6 BPM.

Change in prestimulus level. The data were examined to determine whether either the experimental or control group changed in prestimulus level over trials. An analysis of variance (ANOVA) was performed with the average of the 6 prestimulus seconds as the dependent measure. Data for all Ss on trials 1-27 were included. The results of the analysis are presented in Table 1. A significant groups by trials interaction was obtained ($F = 1.99$; df 26,364; $p = .01$). Figure 4 shows that the prestimulus level increased over trials for the experimental Ss, but did not increase for the control Ss.

In order to better define this result several further analyses were conducted. The prestimulus level during the first block of conditioning trials (block 2, trials 4-6) was compared with the last block of conditioning trials (block 9, trials 25-27) for both groups in an ANOVA. The group means are shown in Table 2 with the results of the ANOVA. The experimental group rose from a mean of 158 BPM on block 2 to a mean of 168 BPM on block 9. The control group showed little change with a group mean of 153 BPM on block 2 and 154 BPM on block 9. However, the two groups did not differ significantly due to tremendous variability between Ss. But, when intersubject variability was eliminated in a second ANOVA by comparing group averages on

each conditioning trial during the first half of the experiment (trials 4-15) with group averages during the second half (trials 16-27), the experimental and control groups were found to differ greatly. Table 3 shows that the groups differed significantly ($F = 30.1$; df 1,44; $p = .001$) and the groups by trials interaction resulted in an F of 15.6 (df 1,44; $p = .001$).

In a final analysis the last block of conditioning trials (block 9) was tested against block 10 (the average of the three extinction trials). As shown in Table 4, the group mean for experimental Ss on block 9 was 168 BPM and decreased to 164 BPM on block 10. Control Ss had a group mean of 150 BPM on block 9 which increased to 152 BPM on block 10. Although intersubject variability was not great for this ANOVA, the two groups were not found to differ.

In summary, the analyses on prestimulus level indicate that over conditioning trials the prestimulus level rose for Ss in the experimental group, but not for Ss in the control group. No significant change was found between the last conditioning trials and the extinction trials.

Change in CR during conditioning trials. The conditioned response (CR) was measured during the first 6 seconds of tone presentation. (On the seventh second the experimental Ss were offered the bottle.) The HR response during these 6 seconds was analyzed for any change over trials. Three ANOVAs were performed comparing the HR response on trial block 2 (the first 3 conditioning trials) with the responses made

later in conditioning (blocks 4, 6, and 9). In addition to the ANOVA for each comparison, linear and quadratic trends over seconds were also tested. The trend tests were used because they were not influenced by the differences in base level already known to be present and were sensitive to variations in the shape of the HR response.

Figure 5 shows the HR response for the experimental and control groups on block 2 and block 4. For both groups the response was a deceleration on both trial blocks. The ANOVA and trend tests are presented in Tables 5A and 5B. A significant groups by trials by seconds interaction was found ($F = 2.53$; df 5,70; $p = .05$). As can be seen in Figure 5 the experimental group showed a pronounced HR deceleration to the tone on block 2 and a smaller HR response on block 4. The control group showed exactly the opposite pattern with a small HR response on block 2 and a large HR response on block 4. The decrease in response from block 2 to block 4 shown by the experimental group is an expected result due to habituation. But, the increase in response from block 2 to block 4 shown by the control group is an unexpected finding.

Further examination of the data revealed that a long response latency for the control group on block 2 resulted in a loss of response as measured by the statistical analyses. Since HR was analyzed only during the first 6 seconds of tone presentation no HR deceleration which occurred after the 6th second was considered in the statistical tests. On block 2 the total HR

response to the tone for the control group was a 6 BPM deceleration. However, only a 1.5 BPM deceleration occurred during the first 6 seconds of tone presentation. Consequently in Figure 5 and on the statistical tests, where only the first 6 seconds of the HR response was considered, the response of the control group on block 2 was represented as being very small. The full response can be seen in Figure 3. On block 4 the full HR response to the tone for the control group was again a 6 BPM deceleration. However, on this trial block all of the responses occurred during the first 6 seconds of tone presentation. Thus, by observing HR only during the first 6 seconds of tone presentation the HR response to the tone on block 4 is made to appear much larger than the HR response on block 2, resulting in the significant groups by trials by seconds interaction already observed.

The HR responses for both groups on trial blocks 2 and 6 are shown in Figure 6. The control group continued to show strong HR deceleration to the tone on block 6. The experimental group, however, showed a mild acceleration. Nevertheless, the ANOVA and trend tests given in Tables 6A and 6B did not find these differences to be significant.

Figure 7 compares the HR response for the two groups on trial blocks 2 and 9. On block 9 the experimental group showed an even greater acceleration to the tone than previously, while the control group continued to show a mild HR deceleration. But, the results of the ANOVA and trend tests (Tables 7A and 7B)

failed to find these differences significant.

The last analyses dealing with the change in the HR response to the tone over conditioning trials considered all 4 of the trial blocks (blocks 2, 4, 6, and 9) at once. An ANOVA and trend tests were conducted for the experimental and control groups separately. The results for the experimental group are given in Tables 8A and 8B. Although the data suggest a change in HR response to the tone over conditioning trials, the results did not meet the traditional $p = .05$ significance level ($F = 1.55$; df 15,105; $p = .10$). The ANOVA and trend tests for the control group are presented in Tables 9A and 9B. Although a significant seconds effect was obtained ($F = 27$; df 5,35; $p = .05$), there is no evidence of any consistent change in the HR response to the tone for this group.

Thus, the statistical tests conducted provided no evidence of the development of a CR to the tone during conditioning trials. Although the experimental group appeared to be showing more HR acceleration as conditioning proceeded, this trend was not found to be significant.

Change in CR during extinction trials. It was predicted that, during extinction trials when the tone no longer signaled presentation of the bottle, the experimental Ss would respond differently to tone presentation than they had when they had expected the bottle. Trials 27 and 28 were averaged together to form a two trial block (block 27-28) measuring the HR response when the bottle was expected. On trial 28, during the first 6

seconds of tone presentation, the experimental Ss would be expecting the bottle to soon be offered, although it was not offered on this trial. Thus, trials 27 and 28 were the last two trials on which the experimental infants would expect the tone to be followed by the bottle, if this connection had been learned. Trials 29 and 30 formed a second 2 trial block (block 29-30) measuring HR when the tone no longer predicted the bottle. On trial 29 the tone would not have the same predictive value as previously, since it had not been followed by the bottle on trial 28. And, by trial 30 the expectancy that the tone would be followed by the bottle would be even lower. Thus, trials 29 and 30 provided a measure of any new response to the tone resulting from its loss of predictive value.

Figure 8 shows the average HR of the experimental and control groups on these blocks. The experimental group showed a steady acceleration on block 27-28, but on block 29-30 deceleration occurred following the 4th second. The control group showed deceleration on both trial blocks. An ANOVA was performed comparing HR during the first 6 seconds of tone presentation for the two groups on these blocks (Table 10A). The groups by trial blocks by seconds interaction was highly significant ($F = 5.46$; df 5,60; $p = .001$) reflecting large differences between the groups during extinction trials. A groups by seconds interaction was also obtained ($F = 2.49$; df 5,60; $p = .05$). Trend tests were performed (Table 10B) and a significant groups by trial blocks linear trend over seconds ($F = 8.66$; df 1,6; $p =$

.05) which substantiated the results of the ANOVA. Graphs of these data (Figures 9 and 10) revealed that responses during extinction trials were cut short by analyzing only the first 6 seconds. During conditioning trials it was not appropriate to analyze beyond 6 seconds due to HR acceleration during UCS presentation in the experimental group. During extinction the groups received the same procedures throughout the 14 seconds of tone. Thus, the groups were compared for the entire 14 second tone period on trials 28 and 29, the first two extinction trials. A groups by trials by seconds interaction was significant (Table 11, $F = 2.21$; df 13,156; $p = .01$). As can be seen in Figures 9 and 10, the experimental group showed a fairly flat curve on trial 28, but deceleration appeared on trial 29 that peaked around seconds 6-7, approximately when Ss had been receiving the bottle. Follow-up analyses of this interaction showed the experimental group, when tested alone, maintained the trials by seconds interaction. The results of the ANOVA are presented in Table 12A ($F = 2.44$; df 13,78; $p = .01$). The decelerative response was further substantiated by the quadratic trend over seconds (Table 12B, $F = 8.65$; df 1,6; $p = .05$) and the greater downward trend of trial 29 was reflected by a trials by seconds interaction on the linear component of seconds ($F = 5.63$; df 1,6; $p = .06$). The control group showed no reliable effects on these trials (Tables 13A and 13B).

In addition, for each group, trial 29 was tested separately for a seconds effect which would indicate a significant

deceleratory trend. These results, which appear in Tables 14A and 14B, showed a reliable deceleration in the experimental group ($F = 3.20$; df 13,78; $p = .01$) which was verified by the quadratic trend over seconds ($F = 5.96$; df 1,6; $p = .06$). The control group did not show a reliable seconds effect.

Trial 28 was also compared with trial 30 in order to determine whether or not the deceleratory response was maintained. Tables 16A and 16B present these results. No significant differences were found, indicating that the effect was ephemeral.

C H A P T E R V

DISCUSSION

The results do not clearly indicate whether or not conditioning was established. The groups were found to differ with respect to change in prestimulus level over trials and on one extinction trial. A trend was noted for a change in the experimental group's response to the tone during conditioning trials, although the traditional .05 significance level was not obtained. Each of these findings will be considered in further detail in an effort to explain the results.

The analyses conducted pertaining to the change in prestimulus level over trials indicated that the groups differed significantly, but that there was tremendous individual variability. A closer examination of the data showed that in the experimental group HR rose during conditioning trials for 4 Ss and fell or remained the same for 4 Ss. The average increase in HR was 22 BPM. For 3 of the 4 experimental Ss whose HR increased over trials, notes taken during the conditioning process suggested that the HR rise was produced by crying. Only 2 experimental Ss showed a decline in HR level of 5 BPM or more. One of these infants fussed during the early trials and become quiet by the end of the procedure. The other infant had been entirely breast fed and experienced difficulty at first in accepting the bottle of glucose. Three control Ss showed increases in HR level over trials and 5 Ss either decreased in HR or showed no change. The average rise in HR was 12 BPM.

one of these 3 infants cried during the experiment and only one fussed. Of the 5 infants who did not show a rise in HR only two showed a decrease of 5 BPM or more. One infant, who showed an HR decrease of 40 BPM over trials, was extremely active and fussy at the beginning of the procedure, but had quieted down by the end. The second infant, who decreased 22 BPM, was also very fussy initially, but was almost asleep at the end of the experiment.

These observations suggest that change in prestimulus HR level over conditioning trials was more a function of random variables than a result of the conditioning process. The rise in HR found in the experimental group appears to be due to crying in a few infants. A comparison of feeding schedules showed that these infants had been fed more recently than most of the other infants and were therefore probably less interested in the glucose.

Although statistical significance was not obtained there was some suggestion of an acceleratory CR developing in the experimental group over trials. This trend is unusual since most HR conditioning studies with adults have found the CR to be deceleratory (see Page 13). The possibility that the CR in young infants might be an HR acceleration was considered. It has already been pointed out (Page 10) that the HR response to most stimuli is an HR acceleration in newborns and only gradually becomes a deceleration by two months of age. Thus, it was possible that some of the infants were within a transition

period between consistent HR deceleration and HR acceleration. However, evidence suggested that this hypothesis was unlikely. For one thing, there was no tendency for younger Ss to show more HR acceleration as would be expected if these infants were in a transition period. Secondly, in Clifton's (1970) study where the infants were much younger (6 to 12 weeks of age), there was no evidence of HR acceleration developing as a CR. In fact, the only suggestion of conditioning in that study was a tendency for the experimental group to maintain a deceleratory response to the tone, as would be expected from adult HR conditioning studies. Finally, the HR acceleration observed in the present study did not appear to be a consistent response developed over trials, but rather occurred on some trials and not on others.

Thus, the acceleratory tendency in the experimental group does not suggest the presence of conditioning. The HR acceleration may have resulted from satiation or boredom which could have caused the tone presentation to become a noxious event eliciting a DR, instead of an informative event producing an OR.

Statistical tests conducted on extinction trials revealed that the experimental and control groups differed in the HR response produced on trial 29. On the previous trial Ss had expected the bottle during the first 6 seconds of tone presentation, but the bottle had not been offered. Thus, trial 29 was the first extinction trial on which Ss could be aware that the tone no longer signaled the offering of the bottle. On

this trial, 5 of the 7 experimental Ss for whom extinction data was obtained showed an HR deceleration of 8 BPM or more. Of the two Ss in this group who failed to show deceleration, one was crying, and the other did not receive the same extinction procedure (see S #13, Appendix B). All of the experimental Ss who showed HR deceleration reached the peak of the deceleratory response between 6 and 9 seconds after tone onset. This interval closely coincides with the time at which the bottle was usually offered. Three of the control Ss also showed HR deceleration on trial 29. In two instances the peak deceleration occurred 3 seconds after tone onset, and in the other instance 11 seconds after tone onset.

The large deceleratory response observed in the experimental group on trial 29 did not reappear on trial 30. Although there was some indication of HR deceleration in the experimental group during the first few seconds of this trial (see Figure 10), control Ss also decelerated to the tone at this time. Thus, the only evidence of conditioning obtained in the present study was the differential response of the 2 groups which occurred on trial 29. But, since trial 29 was the crucial trial and the response obtained was highly consistent and in accordance with expectations, there is a strong possibility that conditioning may have, in fact, occurred. It is quite possible that by the last extinction trial, after two trials in which the tone was presented without the bottle, the discrepancy between the expectation of the bottle and its non-appearance had dissipated. Unfortunately,

the lack of other supporting evidence prevents any definitive conclusions from being drawn.

Several explanations can be offered to account for the absence of clear conditioning in the present experiment. First of all, it could be the case that HR conditioning is not possible with infants of this age. This conclusion appears unlikely, however, in that 3 month old infants are clearly conditionable (Brackbill & Fitzgerald, 1969) and successful HR conditioning has been reported for premature infants (Polikanina, 1961). A more likely hypothesis is that external factors introduced variability into the data, obscuring the effects of any conditioning which did occur. Such variability could have resulted from any one of several conditions. To begin with, it has already been suggested that satiation may have occurred in some Ss. Although an effort was made to test infants at least an hour after their last feeding, this was not always possible. A second contributor to the lack of results may have been the wide variability which occurs in the activity level of the infants during the experimental procedure. The infants frequently became fussy, sleepy, or sometimes cried. Since HR varies with these different activities, it is difficult to determine whether any change observed in HR is a function of learning, activity level, or a combination of the two. Perhaps, a stricter criterion for discarding the data of Ss who show a marked change in state was needed. In the present procedure Ss were eliminated only when they did not accept the bottle for 3 consecutive times

or cried through 3 tone presentations. Consequently several fussy infants remained in the experiment, adding a great deal of variability to the data.

Another factor affecting conditioning may have been the stimulus used as CS. The present experiment utilized an auditory CS of 14 seconds duration which remained on while the bottle was offered (experimental group). Such a long auditory stimulus may have become noxious to the infants and increased fussiness. Furthermore, under this procedure the presentation of the bottle was not keyed to either the onset or the offset of the tone. Thus, on trial 28, the first extinction trial, the infants would not know for sure until the tone went off whether or not the bottle was forthcoming. Also, an auditory stimulus may not have been the most effective CS. Brackbill & Fitzgerald (1969) found that change in pupillary diameter, another autonomic response, could not be conditioned to an auditory CS, but could be conditioned to a temporal CS.

Thus, several implications for future research in infant HR conditioning are evident. First of all, if an appetitive UCS is used, the experimenter should attempt to maximize its reinforcing value. One possibility is to use formula as the UCS and schedule the experiment in place of a regular feeding. A second type of UCS which has recently been used is vestibular stimulation. Malcuit and Clifton (1971, unpublished data) are now conducting a study in which the UCS is a rocking of the infant's crib. A second implication is the use of a larger

number of Ss in each group, but at the same time establishing a strict criterion of eliminating Ss who are extremely active, fussy or crying. In this manner, a better measure of the learning process might be obtained and some of the variability resulting from change in state could be eliminated. Lastly, if an auditory stimulus is employed as the CS, it should be of a shorter duration with its offset timed to coincide with the onset of the UCS. Other types of CSs such as time should also be considered in view of the Brackbill & Fitzgerald (1969) results.

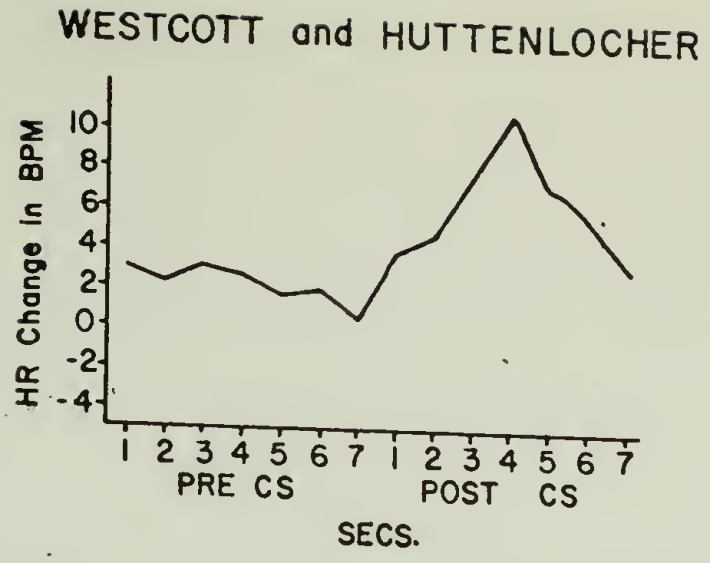
In summary, the present study was conducted in order to determine whether or not HR could be conditioned in 3 month old infants. The conditionability of HR as an autonomic response was viewed in terms of Brackbill and Fitzgerald's (1969) hypothesis regarding differential conditioning results as a function of measuring an autonomic rather than a somatic response. The relative scarcity of conditioning studies measuring autonomic responses prompted a study by Clifton (1970, unpublished data) which yielded inconclusive results. The present study was designed as a replication of the Clifton study with several modifications.

The experiment included two groups of 8 Ss each. The CS was a 14 second 70 dB tone and the UCS a bottle of glucose. Experimental Ss heard the tone for 6 seconds after which the bottle and tone were presented simultaneously. Control Ss received a Rescorla control in which the CS and UCS sometimes

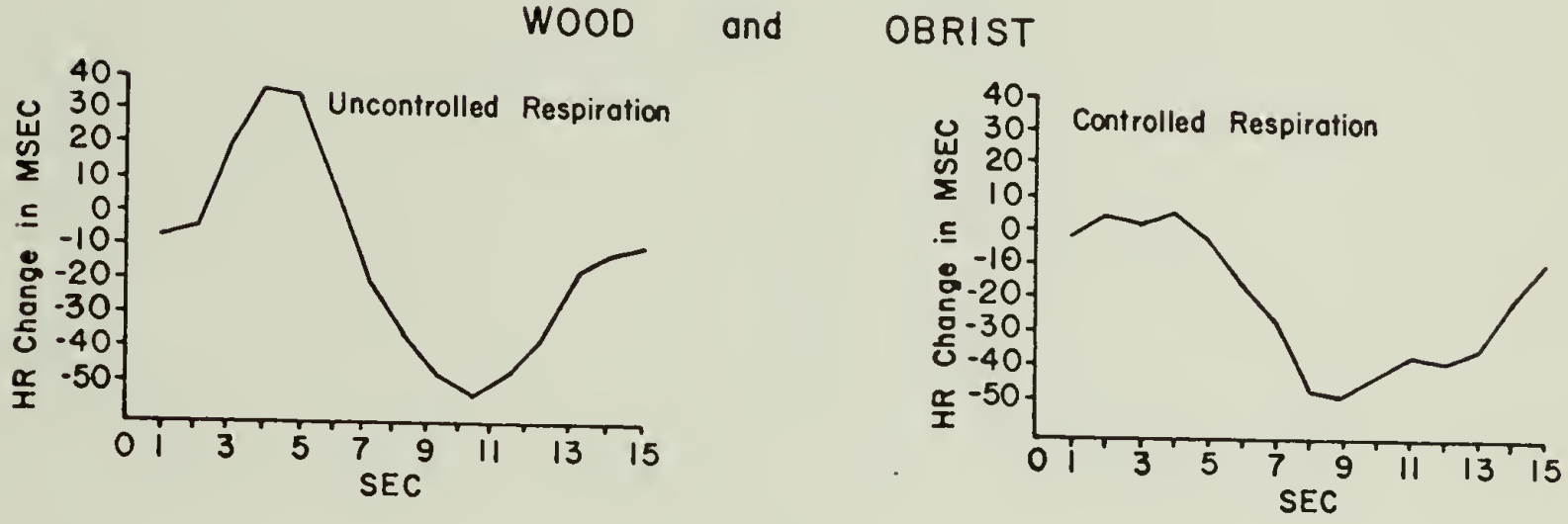
occurred together and sometimes did not. The results gave unsubstantial evidence of conditioning. No consistent CR was developed and the groups differed on only one of the extinction trials. Several conditions such as satiation, duration and type of CS, and change in state were considered as possible sources of HR variability which may have obscured the results. Some suggestions were made to modify future infant HR conditioning studies.

A P P E N D I X A

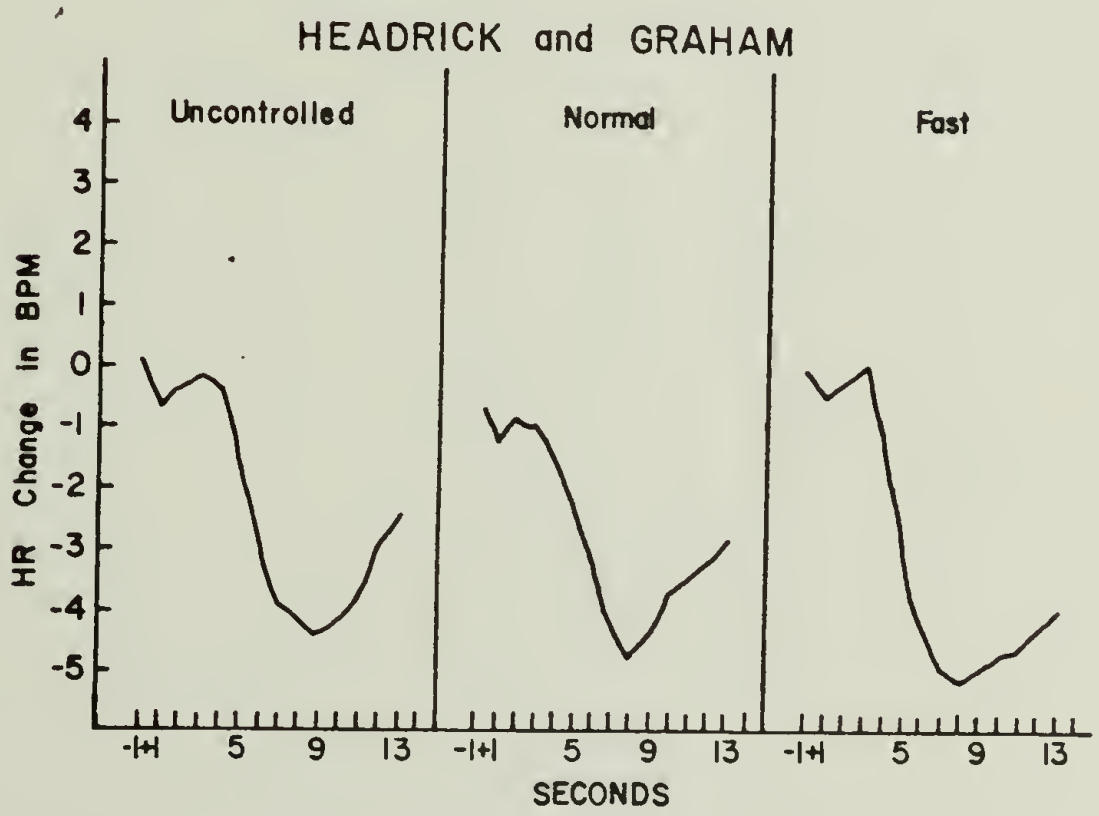
GRAPHS



A. The HR response observed by Westcott and Huttenlocher during the last conditioning trials.



B. The HR responses observed by Wood and Obrist on test trials.



C. The HR responses observed by Headrick and Graham on test trials.

INITIAL RESPONSE TO THE BOTTLE

(AVERAGE OF TRIALS 1-3)

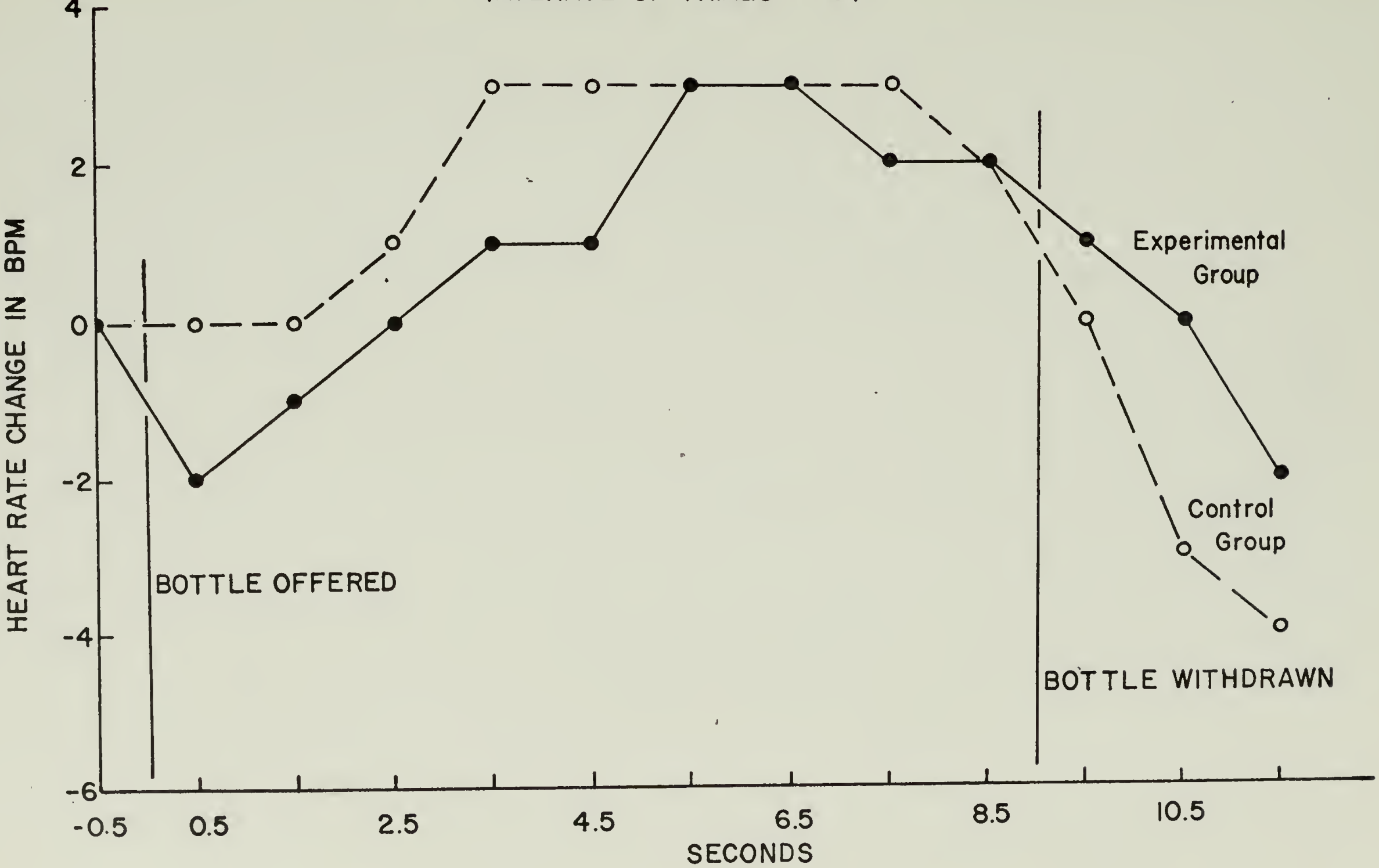


FIGURE 2

(GROUP AVERAGE OF TRIALS 4-6)

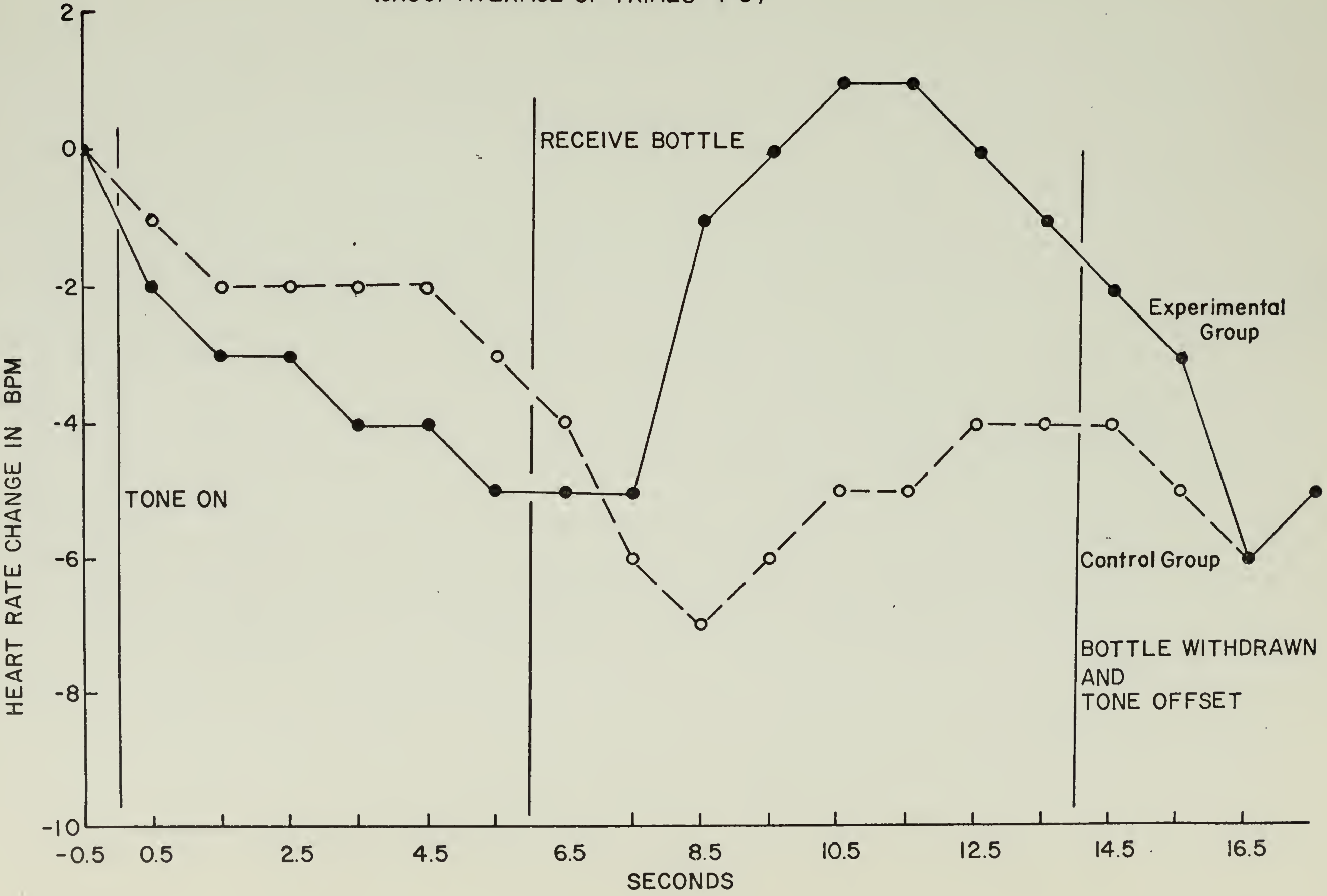
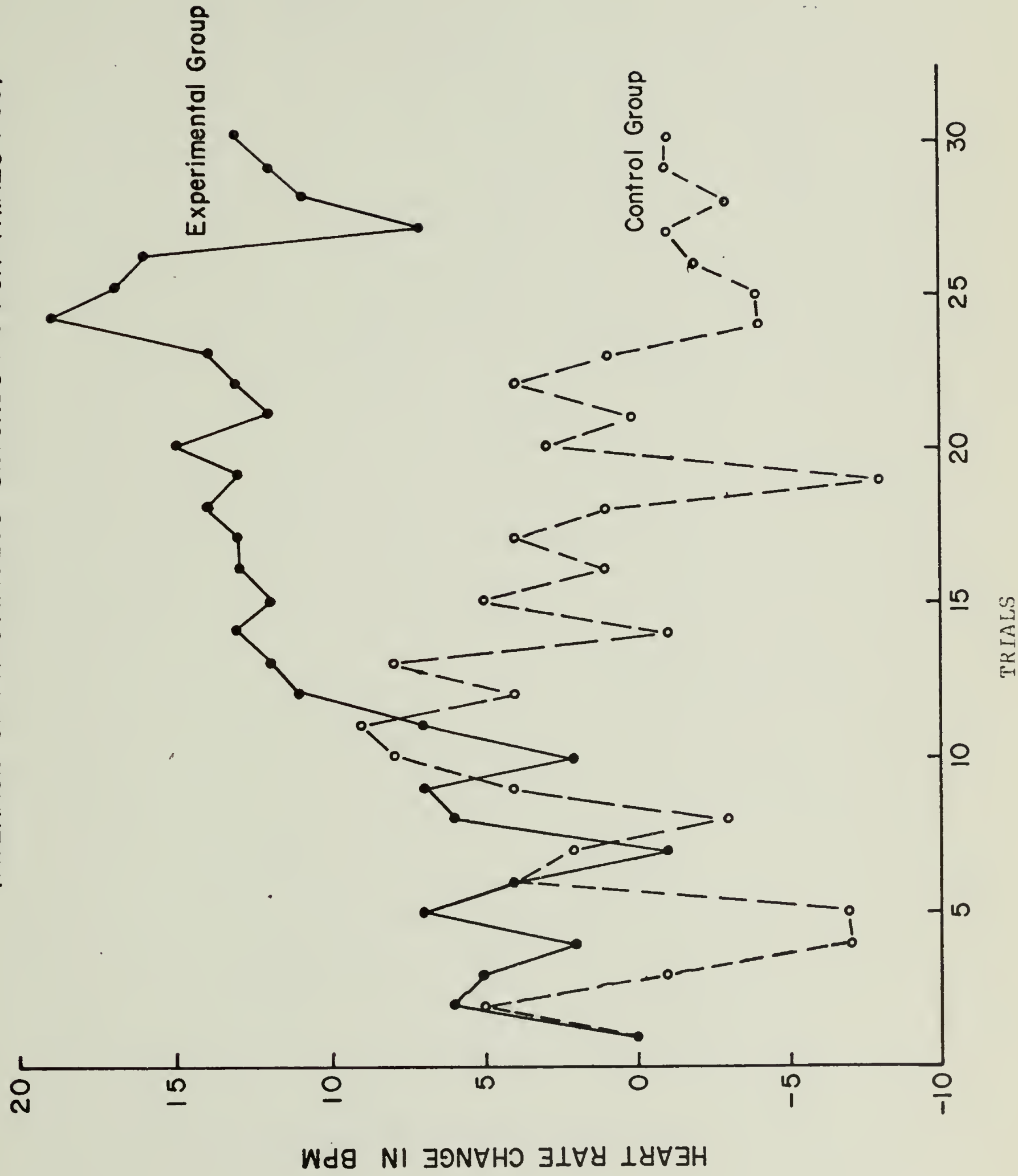


FIGURE 4


PRESTIMULUS HEART RATE
(AVERAGE OF PRESTIMULUS SECONDS 1-6 FOR TRIALS 1-30)
Experimental Group
Control Group
HEART RATE CHANGE IN BPM
20
15
10
5
0
-5
-10
5
10
15
20
25
30
TRIALS

CHANGE IN CR. – BLOCK 2 vs BLOCK 4

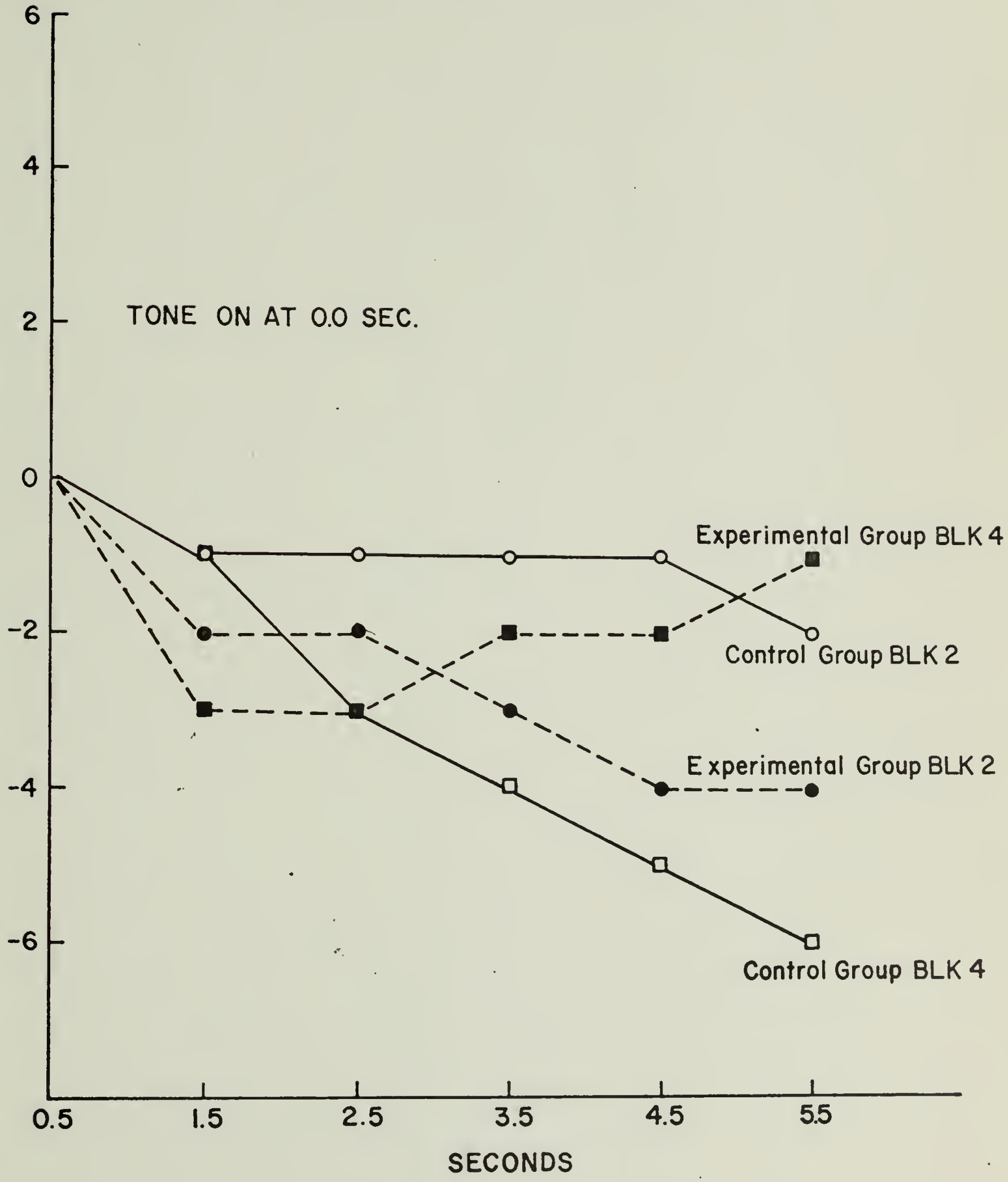
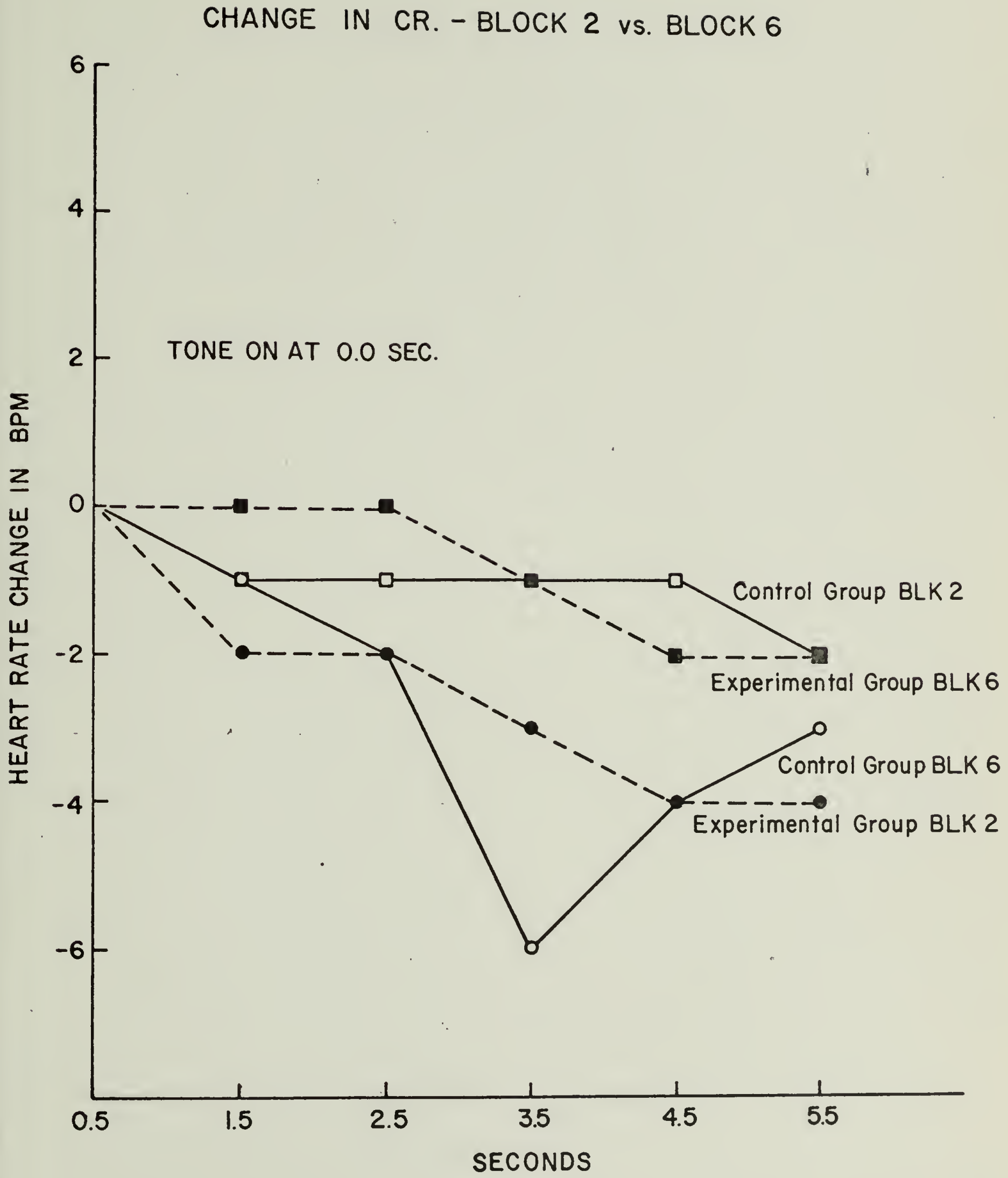
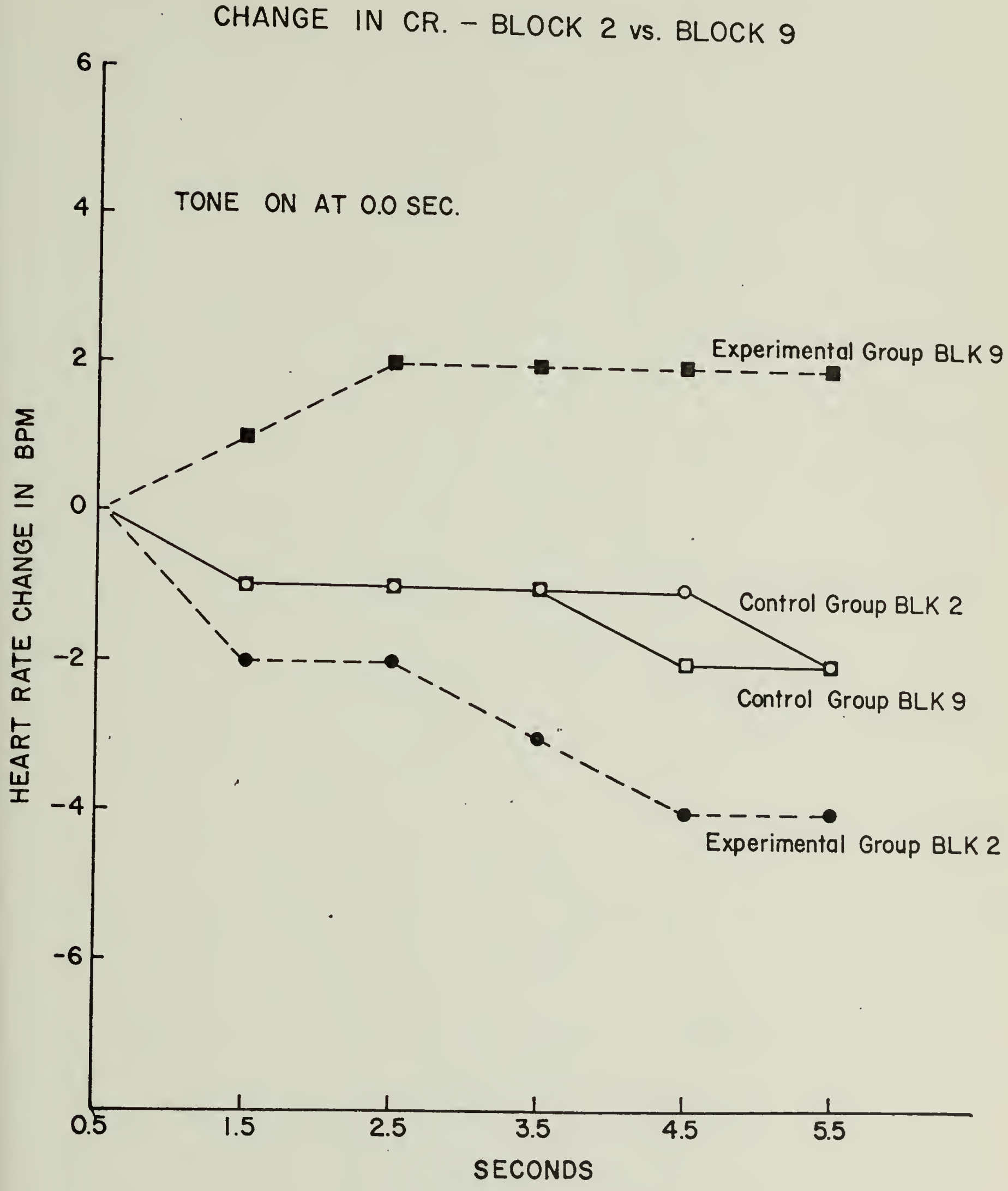
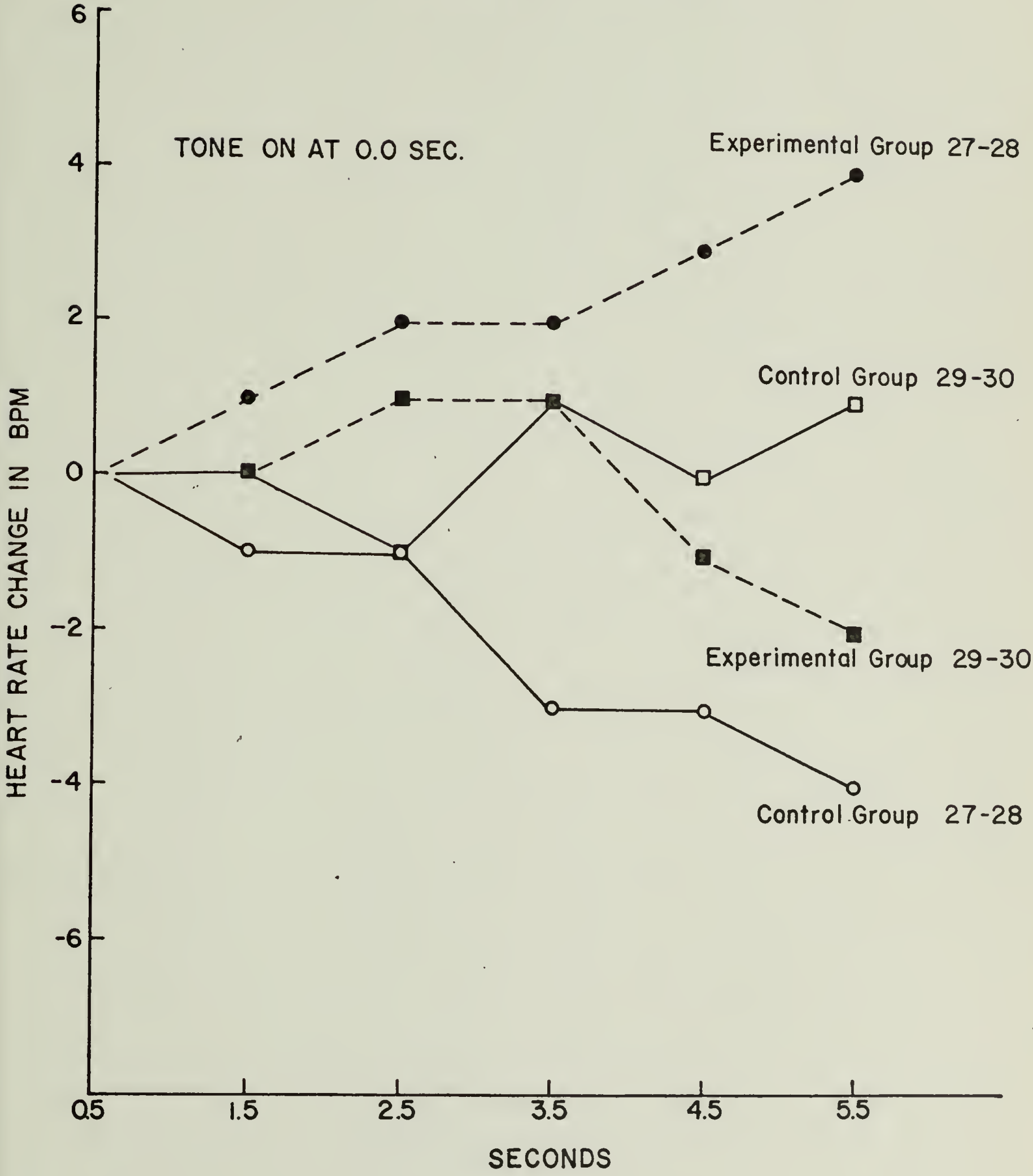


FIGURE 6


CHANGE IN CR. - BLOCK 2 vs. BLOCK 6
6
4
2
0
-2
-4
-6
HEART RATE CHANGE IN BPM
TONE ON AT 0.0 SEC.
Control Group BLK 2
Experimental Group BLK 6
Control Group BLK 6
Experimental Group BLK 2
0.5
1.5
2.5
3.5
4.5
5.5
SECONDS


CHANGE IN CR. – BLOCK 2 vs. BLOCK 9
TONE ON AT 0.0 SEC.
Experimental Group BLK 9
Control Group BLK 2
Control Group BLK 9
Experimental Group BLK 2
HEART RATE CHANGE IN BPM
6
4
2
0
-2
-4
-6
0.5
1.5
2.5
3.5
4.5
5.5
SECONDS

RESPONSE ON EXTINCTION TRIALS
BLOCK 27-28 vs. BLOCK 29-30



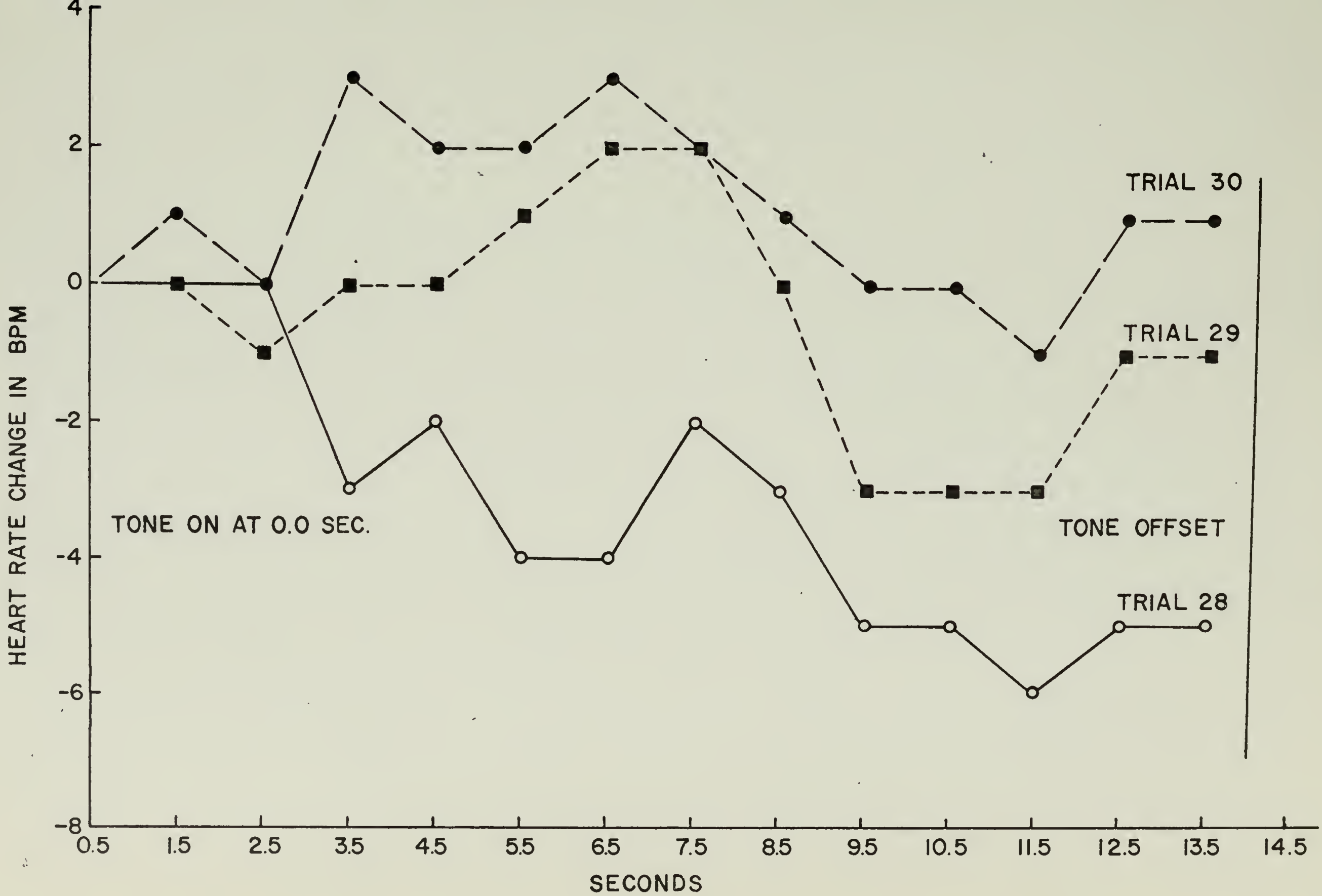
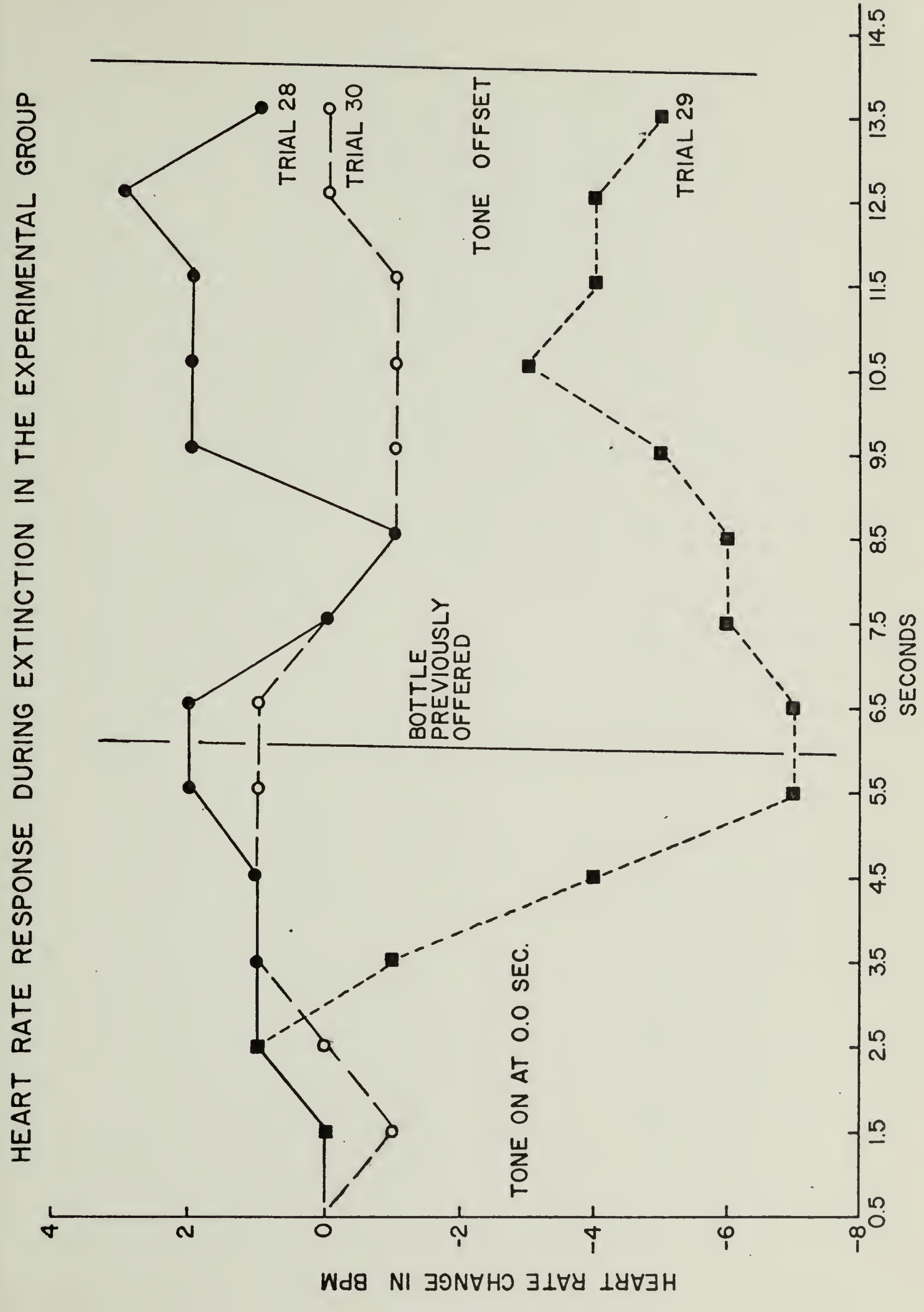

HEART RATE CHANGE IN BPM
4
2
0
-2
-4
-6
-8
TRIAL 30
TRIAL 29
TRIAL 28
TONE ON AT 0.0 SEC.
TONE OFFSET
0.5
1.5
2.5
3.5
4.5
5.5
6.5
7.5
8.5
9.5
10.5
11.5
12.5
13.5
14.5
SECONDS

FIGURE 10


HEART RATE RESPONSE DURING EXTINCTION IN THE EXPERIMENTAL GROUP
HEART RATE CHANGE IN BPM
4
2
0
-2
-4
-6
-8
0.5
1.5
2.5
3.5
4.5
5.5
6.5
7.5
8.5
9.5
10.5
11.5
12.5
13.5
14.5
SECONDS
TONE ON AT 0.0 SEC.
BOTTLE PREVIOUSLY OFFERED
TONE OFFSET
TRIAL 28
TRIAL 30
TRIAL 29

A P P E N D I X B

STATISTICAL TABLES

TABLE I

Analysis of Variance for Change in Prestimulus HR (Average of Six Prestimulus Seconds) Trials 1-27

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
G	36889.03	1	36889.03	1.1+
T	20903.12	26	803.96	1.1+
S[G]	468798.51	14	33485.60	
GT	36492.71	26	1403.56	1.99*
TS[G]	255780.23	364	702.69	

G = groups (exp. vs. control) * significant at .05
T = trials (1.27) + not significant
S = subjects (8 per group)

TABLE II

Analysis of Variance for Change in Prestimulus HR (Average of Six Prestimulus Seconds) Block 2 vs. Block 9

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
G	639.03	1	639.03	2.6+
B	195.03	1	195.03	1.3+
S[G]	3412.18	14	243.72	
GB	124.03	1	124.03	
BS[G]	2141.43	14	152.95	

G = groups (exp. vs. control)
B = trial blocks (2 vs. 9)
S = subjects (8 per group)

+ not significant

	Cell Means	
	2	9
Experimental	157.62	166.50
Control	152.62	153.62

TABLE III

Analysis of Variance for Change in Prestimulus HR
Group Average of Six Prestimulus Seconds
Trials 3-15 vs. Trials 16-17

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
G	513.52	1	513.52	30.1*
B	63.02	1	63.02	3.5+
GB	266.02	1	266.02	15.6*
T(GB)	782.25	44	17.77	

G = groups (exp. vs. control)
B = trial blocks (trials 3-15 vs. trials 16-27)
T = trials (12 per trial block)

*significant at .001
+not significant

Cell Means

	3-15	16-27
Experimental	159.83	166.83
Control	158.00	155.58

TABLE IV

Analysis of Variance for Change in Prestimulus HR
Average of Six Prestimulus Seconds
Trial Block 9 vs. Trial Block 10

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
G	1305.37	1	1305.37	2.13
B	2.04	1	2.04	- +
S[G]	6136.08	10	613.60	
GB	51.04	1	51.04	1.9
SB[G]	269.41	10	26.94	+

G = groups (exp. vs. control)
B = trial blocks (9 vs. 10)
S = subjects (6 per group)

+ not significant

	Cell Means	
	9	10
Experimental	167.66	164.16
Control	150.00	152.33

TABLE VA

Analysis of Variance for Change in CR
Trial Block 2 vs. Trial Block 4

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
G	12.00	1	12.00	
B	2898.52	1	2898.52	6.52+
P	201.91	5	40.38	3.01+
S(G)	42084.25	14	3006.01	
GB	46.02	1	46.02	
GP	30.25	5	6.05	
BP	8.85	5	1.77	
SB(G)	6213.45	14	443.81	
SP(G)	932.50	70	13.32	
GBP	117.60	5	23.52	2.53+
SBP(G)	646.54	70	9.23	

G = groups (exp. vs. control)
B = trial blocks (2 vs. 4)
P = post-stimulus seconds (1-6)
S = subjects (8 per group)

+ significant at .05

Cell Means

Seconds	1	2	3	4	5	6	
Experimental	155.87	154.12	153.74	153.12	152.00	152.12	(2)
	161.75	159.75	159.00	159.75	160.50	161.00	(4)

TABLE VA (cont'd)

Cell Means						
Seconds	1	2	3	4	5	6
Control	153.12	151.75	152.00	151.75	152.12	151.37 (2)
	163.87	162.87	161.37	159.75	159.00	157.75 (4)

TABLE VB

Change in CR - Trial Block 2 vs. Trial Block 4

Trend Tests

	Linear				Quadratic			
Source	SS	DF	MS	F	SS	DF	MS	F
P	174.94	1	174.94	5.20+	21.78	1	21.78	1.48+
PB	3.45	1	3.45	-	2.50	1	2.50	-
PG	18.21	1	18.21	-	9.05	1	9.05	-
PS(G)	338.33	7	35.35	-	103.30	7	14.75	-
PBG	106.31	1	106.31	3.01+	4.66	1	4.66	-
PSB(G)	247.49	7	35.35	-	57.16	7	8.16	-

B = trial Blocks (Block 2 vs. 4)
S = subjects (8 per group)
G = groups (experimental vs. control)
P = post-stimulus seconds (1-6)

+ not significant

TABLE VIA

Analysis of Variance for Change in CR
Trial Block 2 vs. Trial Block 6

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
G	2472.50	1	2472.50	
B	2303.25	1	2303.25	2.50+
P	188.46	5	37.69	3.20*
S[G]	29982.61	14	2141.61	
GB	1558.38	1	1558.38	
GP	30.83	5	6.16	
BP	27.33	5	5.46	
SB[G]	12873.78	14	919.55	
SP[G]	823.44	70	11.76	
GBP	62.96	5	12.59	1.1 +
SBP[G]	755.78	70	10.79	

G = groups (exp. vs, control)
B = blocks (2 vs. 6)
P = post-stimulus seconds (1-5)
S = subjects (8 per group)

* significant at .05
\+ not significant

Cell Means

Seconds	1	2	3	4	5	6	
Experimental	155.87	154.12	153.75	153.12	152.00	152.12	(2)
	166.62	167.00	166.75	166.37	165.12	164.87	(6)

TABLE VIA (cont'd)

Cell Means							
Seconds	1	2	3	4	5	6	
Control	153.12	151.75	152.00	151.75	152.12	151.37	(2)
	155.75	154.76	154.12	150.12	151.50	153.12	(6)

TABLE VIB

Change in CR - Trial Block 2 vs. Trial Block 6

Trend Tests

	Linear				Quadratic			
Source	SS	DF	MS	F	SS	DF	MS	F
P	163.40	1	163.40	6.32*	13.57	1	13.57	-
PB	2.00	1	2.00	-	0.35	1	0.35	-
PG	0.82	1	0.82	-	15.33	1	15.33	1.1+
PS(G)	181.06	7	25.87	-	100.99	7	14.42	-
PBG	26.36	1	26.36	-	22.69	1	22.69	1.0+
PBS(G)	269.44	7	38.49	-	108.72	7	22.67	-

B = trial blocks (2 vs. 6)
S = subjects (8 per group)
G = groups (experimental vs. control)
P = post-stimulus seconds (1-6)

\+ not significant
* significant at .05

TABLE VIIA

Analysis of Variance for Change in CR
Trial Block 2 vs. Trial Block 9

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
G	1496.33	1	1496.33	
B	2836.68	1	2836.68	2.52+
P	39.66	5	7.93	
S(G)	22318.08	14	1594.14	
GB	808.52	1	808.52	
GP	7.16	5	1.43	
BP	35.81	5	7.16	
SB(G)	15739.29	14	1124.23	
SP(G)	529.66	70	7.56	
GBP	67.22	5	13.44	1.52+
SBP(G)	619.45	70	8.84	

G = groups (exp. vs. control)
B = trial blocks (2 vs. 9)
P = post-stimulus seconds (1-6)
S = subjects (8 per group)

\+ not significant

Cell Means

Seconds	1	2	3	4	5	6	
Experimental	155.87	154.12	153.75	153.12	152.00	152.12	(2)
	164.00	164.75	165.50	165.50	165.87	166.12	(9)

TABLE VIIA (cont'd)

Cell Means						
Seconds	1	2	3	4	5	6
Control	153.12 156.87	151.75 156.12	152.00 155.62	151.75 155.50	152.12 155.00	151.37 (2) 154.50 (9)

TABLE VIIB

Change in CR - Trial Block 2 vs. Trial Block 9

Trend Tests

	Linear				Quadratic			
Source	SS	DF	MS	F	SS	DF	MS	F
P	35.00	1	35.00	1.32+	1.25	1	1.25	-
PB	29.71	1	29.71	1.12+	3.87	1	3.87	-
PG	3.77	1	3.77	-	0.14	1	0.14	-
PS(G)	184.63	7	26.40	-	59.85	7	8.60	-
PBG	63.78	1	63.78	1.90+	1.52	1	1.52	-
PBS(G)	230.07	7	32.9	-	52.50	7	7.50	-

B = trial blocks (2 vs. 9)
S = subjects (8 per group)
G = groups (experimental vs. control)
P = post-stimulus seconds (1-6)

\+ not significant

TABLE VIIIA

Analysis of Variance for Change in CR
for the Experimental Group
Trial Blocks 2, 4, 6 and 9

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
S	17037.22	7	2433.88	
B	4851.39	3	1617.13	
P	27.54	5	5.50	
SB	14072.52	21	670.12	
SP	239.70	35	6.84	
BP	151.79	15	10.11	1.53
SBP	696.29	105	6.63	+

S = subjects (7)
B = trial blocks (2, 4, 6, 9)
P = post-stimulus seconds (1-6)

+ not significant

Cell Means

Seconds						Blocks
1	2	3	4	5	6	
155.87	154.12	153.75	153.12	152.00	152.12	(2)
161.75	159.75	159.00	159.75	160.50	161.00	(4)
166.62	167.00	166.75	166.37	165.12	164.87	(6)
164.00	164.75	165.50	165.50	165.87	166.12	(9)

TABLE VIIIB

Change in CR for the Experimental Group

Trial Blocks 2, 4, 6 and 9

Trend Tests

	Linear				Quadratic			
Source	SS	DF	MS	F	SS	DF	MS	F
P	21.21	1	21.21	1.47+	4.50	1	4.50	-
PB	101.89	3	33.96	1.81+	38.32	3	12.77	1.23+
PS	100.60	7	14.37	-	106.96	7	15.28	-
PBS	392.45	21	18.68	-	217.61	21	10.36	-

B = trial blocks (2, 4, 6, 9)
S = subjects (7)
P = post-stimulus seconds (1-6)

+not significant

TABLE IXA

Analysis of Variance for Change in CR
for the Control Group
Trial Blocks 2, 4, 6 and 9

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
S	54804.11	7	7829.16	
B	2156.55	3	718.85	
P	284.52	5	56.90	2.7*
SB	17373.98	21	827.33	
SP	736.09	35	21.03	
BP	159.53	15	10.63	
SBP	1255.67	105	11.95	

S = subjects (7)
B = trial blocks (2, 4, 6, 9)
P = post-stimulus seconds (1-6)

* significant at .05

Cell Means

Seconds						Blocks
1	2	3	4	5	6	
153.12	151.75	152.00	151.75	152.12	151.37	(2)
163.87	162.87	161.37	159.75	159.00	157.75	(4)
155.75	154.87	154.12	150.12	151.50	153.12	(6)
156.87	156.12	155.62	155.50	155.00	154.50	(9)

TABLE IXB

Change in CR for the Control Group

Trial Blocks 2, 4, 6 and 9

Trend Tests

	Linear				Quadratic			
Source	SS	DF	MS	F	SS	DF	MS	F
P	254.47	1	254.47	2.82+	18.17	1	18.17	1.90+
PB	84.49	3	28.16	-	25.52	3	8.50	-
PS	629.84	7	89.97	-	66.71	7	9.53	-
PBS	702.30	21	33.44	-	454.82	21	21.65	-

B = trial blocks (2, 4, 6, 9)
S = subjects (7)
P = post-stimulus seconds (1-6)

\+ not significant

TABLE XA

Analysis of Variance for Extinction Trials
Trial Block 27-28 vs. Trial Block 29-30

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
G	3584.38	1	3584.38	
B	6.09	1	6.09	
P	10.33	5	2.06	
S[G]	39350.57	12	3279.21	
GB	20.02	1	20.02	
GP	48.40	5	9.68	1.57+
BP	12.40	5	2.48	
SB[G]	1660.38	12	138.36	
SP[G]	369.42	60	6.15	
GBP	203.19	5	40.63	5.46*
SBP[G]	445.90	60	7.43	

G = groups (exp. vs. control)
B = trial block (27-28 vs. 29-30)
P = post-stimulus seconds (1-6)
S = subjects (7)

\+ not significant
* significant at .001 (4.76)

Cell Means

Seconds	1	2	3	4	5	6	
Experimental	159.57	160.57	161.85	162.28	163.14	164.00	(27-28)
	162.28	161.57	162.85	162.71	160.57	159.57	(29-30)

TABLE XA (cont'd)

Cell Means							
Seconds	1	2	3	4	5	6	
Control	153.71	153.42	152.85	151.42	151.14	149.28	(27-28)
	152.85	153.00	151.57	153.57	153.42	153.85	(29-30)

TABLE X8

Extinction Trials - Trial Block 27-28 vs.

Trial Block 29-30

Trend Tests

	Linear				Quadratic			
Source	SS	DF	MS	F	SS	DF	MS	F
P	1.83	1	1.83	-	6.53	1	6.53	-
PB	1.71	1	1.71	-	0.00	1	0.00	-
PG	31.88	1	31.88	1.99+	6.32	1	6.32	-
PS(G)	99.86	6	16.64	-	57.17	6	9.53	-
PBG	183.67	1	183.67	8.66*	14.08	1	14.08	1.32+
PBS(G)	127.02	6	21.17	-	65.54	6	10.92	-

B = trial blocks (27-28 vs. 29-30)
S = subjects (7 per group)
G = groups (experimental vs. control)
P = post-stimulus seconds (1-6)

\+ not significant
* significant at .05

TABLE XI

Analysis of Variance for Extinction Trials
Trial 28 vs. Trial 29

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
G	16653.12	1	16653.12	
T	565.92	1	565.92	
P	364.53	13	28.04	
S[G]	102255.90	12	8521.32	
GT	2545.92	1	2545.92	3.30+
GP	422.26	13	32.48	1.30
TP	68.75	13	5.28	
ST[G]	9245.47	12	770.45	
SP[G]	3877.37	156	24.85	*
GTP	529.61	13	40.73	2.21
STP[G]	2863.80	156	18.35	

G = groups (exp. vs. control)
T = trials (28 vs. 29)
P = post-stimulus seconds (1-14)
S - subjects (7)

\+ not significant
* significant at .01

TABLE XI (cont'd)

Cell Means								
Seconds	1	2	3	4	5	6	7	
Experimental	162.71	163.28	163.71	163.85	163.71	164.57	164.57	(28)
	165.28	164.71	166.14	164.28	160.71	157.71	157.71	(29)
Seconds	8	9	10	11	12	13	14	
Experimental	163.00	162.42	164.57	165.14	165.14	165.57	163.85	(28)
	158.85	159.14	159.85	161.71	161.42	161.28	159.57	(29)
Seconds	1	2	3	4	5	6	7	
Control	148.85	149.00	148.71	146.00	146.71	145.85	145.42	(28)
	153.57	153.57	153.14	153.57	154.00	154.85	155.57	(29)
Seconds	8	9	10	11	12	13	14	
Control	146.85	146.14	144.14	143.57	143.28	143.57	144.14	(28)
	155.85	154.42	151.28	151.28	150.57	152.71	152.85	(29)

TABLE XIIA

Analysis of Variance for Extinction Trials
Trial 28 vs. Trial 29 - Experimental Group

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
S	27339.98	6	6204.99	
T	355.59	1	355.59	
P	345.91	13	26.60	1.66+
ST	3048.19	6	508.03	
SP	1246.86	78	15.98	
TP	450.26	13	34.63	2.44*
STP	1106.94	78	14.19	

S = subjects (7)
T = trials (28 vs. 29)
P = post-stimulus seconds (1-14)

\+ not significant
* significant at .01

Cell Means

Seconds	1	2	3	4	5	6	7	
(Experimental)	162.71	163.28	163.71	163.85	163.71	164.57	164.57	(28)
	165.28	164.71	166.14	164.28	160.71	157.71	157.71	(29)

TABLE XIIA (cont'd)

Seconds	8	9	10	11	12	13	14	
	163.00	162.42	164.57	165.14	165.14	165.57	163.85	(28)
	158.85	159.14	159.85	161.71	161.42	161.28	159.57	(29)

TABLE XIIB

Extinction Trials - Trial 28 vs. 29

Experimental Group

Trend Tests

	Linear				Quadratic			
Source	SS	DF	MS	F	SS	DF	MS	F
P	49.25	1	49.25	-	109.62	1	109.62	8.65**
PT	202.45	1	202.45	5.63*	121.10	1	121.10	2.44+
PS	534.11	6	89.01	-	76.04	6	12.67	-
PTS	215.54	6	35.92	-	297.19	6	49.53	-

P = post-stimulus seconds (1-14)
S = subjects (7)
T = trials (28 vs. 29)

* p = .06
** p = .05
\+ not significant

TABLE XIIIA

Analysis of Variance for Extinction Trials
Trial 28 vs. Trial 29 - Control Group

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
S	65025.91	6	10837.65	
T	2756.25	1	2756.25	2.67+
P	440.88	13	33.91	
ST	6197.28	6	1032.88	
SP	2630.51	78	33.72	
TP	148.10	13	11.39	
STP	1756.85	78	22.52	

S = subjects (7)
T = trials (28 vs. 29)
P = post-stimulus seconds (1-14)

+ not significant

Cell Means

Seconds	1	2	3	4	5	6	7	
(Control)	148.85	149.00	148.71	146.00	146.71	145.85	145.42	(28)
	153.57	153.57	153.14	153.57	154.00	154.85	155.57	(29)

TABLE XIIIA (cont'd)

Seconds	8	9	10	11	12	13	14	
	164.85	146.14	144.14	143.57	143.28	143.57	144.14	(28)
	155.85	154.42	151.28	151.28	150.57	152.71	152.85	(29)

TABLE XIII8

Extinction Trials - Trial 28 vs. 29

Control Group

Trend Tests

	Linear				Quadratic			
Source	SS	DF	MS	F	SS	DF	MS	F
P	280.08	1	280.08	-	5.01	1	5.01	-
PT	60.09	1	60.09	-	33.00	1	33.00	2.29+
PS	1861.74	6	310.29	-	101.52	6	16.92	-
PTS	1396.15	6	232.69	-	86.24	6	14.37	-

P = post-stimulus seconds (1-14)
S = subjects (7)
T - trials (28 vs. 29)

\+ not significant

TABLE XIVA

Analysis of Variance for Extinction Trials
Trial 29 - Experimental Group

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
S	24855.69	6	4142.61	
P	712.90	13	54.83	
SP	1336.59	78	17.13	3.20*

S = subjects (7)
P = post-stimulus seconds (1-14)

* significant at .01

Cell Means							
Seconds	1	2	3	4	5	6	7
(Trial 29)	165.28	164.71	166.14	164.28	160.71	157.71	157.71
Seconds	8	9	10	11	12	13	14
	158.85	159.14	159.85	161.71	161.42	161.28	159.57

TABLE XIVB

Extinction Trials - Trial 29

Experimental Group

Trend Tests

	Linear				Quadratic			
Source	SS	DF	MS	F	SS	DF	MS	F
P	225.68	1	225.68	2.51+	230.58	1	230.58	5.96+
PS	539.39	6	89.89	-	232.14	6	38.69	-

P = post-stimulus seconds (1-14)
S = subjects (7)

\+ not significant
* significant at .06

TABLE XVA

Analysis of Variance for Extinction Trials
Trial 29 - Control Group

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
S	31632.24	6	5272.04	
P	225.03	13	17.31	
SP	1647.75	78	21.12	

S = subjects (7)
P = post-stimulus seconds (1-14)

Cell Means

Seconds	1	2	3	4	5	6	7
(Trial 29)	153.57	153.57	153.14	153.57	154.00	154.85	155.57
Seconds	8	9	10	11	12	13	14
	155.85	154.42	151.28	151.28	150.57	152.71	152.85

TABLE XVB

Extinction Trials - Trial 29

Control Group

Trend Tests

	Linear				Quadratic			
Source	SS	DF	MS	F	SS	DF	MS	F
P	40.35	1	40.35	-	31.86	1	31.86	5.40+
PS	1280.35	6	213.39	-	35.22	6	5.87	-

P = post-stimulus seconds (1-14)
S = subjects (7)

\+ not significant

TABLE XVIA

Analysis of Variance for Extinction Trials
Trial 28 vs. Trial 30 - Experimental Group

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
S	41795.74	6	6965.95	
T	824.51	1	824.51	
P	84.38	13	6.49	
ST	4851.70	6	808.61	
SP	1193.54	78	15.30	
TP	54.77	13	4.21	
STP	771.01	78	9.88	

S = subjects (7)
T = trials (28 vs. 30)
P = post-stimulus seconds (1-14)

Cell Means

Seconds	1	2	3	4	5	6	7
(Experimental)	162.71	163.28	163.71	163.85	163.71	164.57	164.57 (28)
	159.57	158.85	160.00	160.71	160.71	161.14	161.28 (30)

TABLE XVIA (cont'd)

Seconds	8	9	10	11	12	13	14
	163.00	162.42	164.57	165.14	165.14	165.57	163.85 (28)
	159.71	159.28	159.00	159.28	159.42	160.14	159.57 (30)

TABLE XVI B

Analysis of Variance for Extinction Trials
Trial 28 vs. Trial 30 - Control Group

Source	Sum of Squares	Degrees of Freedom	Mean Square	F
S	59314.63	6	9885.77	
T	1806.25	1	1806.25	1.2
P	259.73	13	19.98	
ST	8616.28	6	1436.04	
SP	1779.65	78	22.81	
TP	223.82	13	17.21	
STP	4191.14	78	53.73	

S = subjects (7)
T = trials (28 vs. 30)
P = post-stimulus seconds (1-14)

Cell Means

Seconds	1	2	3	4	5	6	7
(Control)	148.85	149.00	148.71	146.00	146.71	145.85	145.42 (28)
	150.85	152.14	150.71	153.57	152.85	152.85	153.85 (30)

TABLE XVI[B] (cont'd)

Seconds	8	9	10	11	12	13	14	
	146.85	146.14	144.14	143.57	143.28	143.57	144.14	(28)
	152.42	151.71	151.28	150.85	149.85	152.28	152.00	(30)

A P P E N D I X C

DATA CORRECTION

Correction of artifacts in the data and elimination of Ss whose data was incomplete was determined according to the following guidelines.

1. In cases where one or two beats differed from the surrounding beats by 25 bpm or more, the divergent beats were replaced by an average of the surrounding beats.
2. If more than three consecutive seconds were lost on any trial then the other two trials in that trial block were averaged to replace that trial.
3. If more than one trial in a trial block was missing enough data to require averaging it then the S was eliminated.
4. If any of the first three or last three conditioning trials required averaging the S was eliminated.
5. If more than three trials were missing or required averaging the S was eliminated.

Corrections and Irregularities in the Experimental Group

#2 trial 19 averaged
trial 30 averaged

#7 seconds 15-17 averaged on trial 5
second 16 averaged on trial 6
trial 17 averaged

#13 seconds 7-8 averaged on trial 1
a delay occurred between the conditioning trials and the extinction trials due to equipment failure.

#14 second 4 on trial 23 was averaged
no extinction trials were obtained for this S due to equipment failure.

#18 second 3 averaged on trial 3
trial 17 averaged
seconds 3-5 averaged on trial 19

Corrections and Irregularities in the Control Group

#5 second 18 averaged on trial 3
seconds -1, 1, and 18 averaged on trial 11
trial 17 averaged

#9 trial 1 averaged

#15 seconds -1 and 1 averaged on trial 4

#17 trial 21 averaged

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